THE

AMERICAN REVIEW OF TUBERCULOSIS ABSTRACTS

VOLUME LV JUNE, 1947 ABST. NO. 6

Streptomycin in Tuberculous Meningitis.—Report of a case of tuberculous meningitis following hematogenous tuberculosis in a 4-year-old boy treated with streptomycin is given. Miliary infiltrations throughout both lung fields were seen in the admission film. Streptomycin 1.0 g. intramuscularly and 0.01 gm. intrathecally daily was begun on the fifth hospital day; doses were raised to 1.2 g. intramuscularly in divided doses every two hours and 0.1 g. intrathecally daily on the ninth day. Because of red cells in the spinal fluid and a persistent febrile reaction, the drug was stopped after a total of 85.0 g. intramuscularly and 3.0 g. intrathecally had been administered. Chest films six weeks after admission showed clearing of the disseminated lesions. Convulsions, strabismus and bacilli appeared again in the spinal fluid three months after entry and caused streptomycin to be again administered; the child died within the next month. No extrapulmonary source of the bacilli was located antemortem. During administration of the drug, the patient showed an apparent auditory neuritis on the eighteenth day, with a concomitant leucopenia. Septic fever and a miliary skin eruption were also observed. An acute diphtheritic pharyngo-tonsillitis developed during the streptomycin therapy; penicillin produced prompt clearing. Postmortem examination showed healing pulmonary tubercles, miliary splenic tubercles and a caseous parenchymal lesion in the brain directly continuous with a caseous exudate in the meninges.—*Streptomycin in Miliary Tuberculosis with Tuberculous Meningitis, P. K. Bornstein, Quart. Bull. Sea View Hosp., July, 1946, 8: 219.*—(*P. Q. Edwards*)

Streptomycin in Tuberculous Meningitis.—A 15-month-old boy took sick with fever, irritability, restlessness and anorexia. A diagnosis of follicular tonsillitis was made and the child was treated with sulfadiazine and penicillin. After temporary improvement recurrence of the fever and restlessness was observed. Physical examination revealed nuchal rigidity and hyperactive knee jerks. Examination of the spinal fluid gave the following result: sugar, 15 mg.; chlorides, 460 mg.; positive Levinson test, pellicle formation on standing, acid-fast bacilli on direct smear. A chest film showed hilar enlargement, more marked on the right side. A tuberculin patch test was positive. The child received 24,000,000 units of streptomycin intramuscularly and 2,800,000 units intrathecally, over a period of fifty-seven days. The child recovered completely. The probable source case was the maternal grandmother who was treated for pleurisy at a tuberculosis hospital. Guinea pig inoculations with spinal fluid were done only after treatment with streptomycin had been started. They were negative for tuberculosis.—*Tuberculous Meningitis Treated with Streptomycin, L. F. Krafchik, J. A. M. A., October 19, 1946, 132: 875.*—(*H. Abeles*)

Streptomycin and Promin in Rat Tuberculosis.—Rats inoculated with a human strain of tubercle bacilli (A27) were treated with streptomycin and promin, individually and in combination, and the effect of treatment determined by (*a*) direct tissue smears, (*b*) subculturing of lung suspensions, (*c*) subinoculation of lung suspensions in guinea pigs. Treatment with promin alone showed no

beneficial effects. Treatment with streptomycin alone resulted in an average lower colony count than in the controls, while treatment with both drugs appeared to indicate sterilization of 41.6 per cent of the animals and marked decrease and attenuation of persisting viable tubercle bacilli in the remainder.—*Influence of Streptomycin and Promin on Proliferation of Tubercle Bacilli in the Tissues of Albino Rat, M. I. Smith, W. T. McClosky & E. W. Emmart, Proc. Soc. Exper. Biol. & Med., June, 1946, 62: 157.—(F. B. Seibert)*

Inactivation of Streptomycin.—The antibacterial activity of streptomycin can be largely or completely neutralized or antagonized by glucose and certain other sugars, an anaerobic environment, certain sulfhydryl compounds, and ketone reagents. In some cases, the effect can be traced to the acidity produced. In the effect of cysteine, cevitamic acid, and of ketone reagents the inhibition of streptomycin activity may be associated with the blocking of an active grouping in the molecule of the streptomycin.—*The Inactivation of Streptomycin, and Its Practical Applications, W. B. Geiger, S. R. Green & S. A. Waksman, Proc. Soc. Exper. Biol. & Med., February, 1946, 61: 187.—(F. B. Seibert)*

Streptomycin Resistant Strains of Tubercle Bacilli.—Streptomycin resistant human type tubercle bacilli were found to be as virulent for white mice as streptomycin sensitive strains. Infection produced in mice with these streptomycin resistant cultures was not suppressed by treatment of the mice with streptomycin.—*Effect of Streptomycin on Experimental Infections Produced in Mice with Streptomycin Resistant Strains of M. tuberculosis var. Hominis, G. P. Youmans & Elizabeth H. Williston, Proc. Soc. Exper. Biol. & Med., October, 1946, 63: 131.—(F. B. Seibert)*

Absorption and Excretion of Streptomycin.—Streptomycin is rapidly absorbed and excreted following parenteral administration. The rapid disappearance of streptomycin from the blood is largely accounted for by its early appearance in the urine. Approximately 60 to 80 per cent of the drug is excreted in the urine of dogs within a twenty-four-hour period after parenteral administration. Somewhat smaller amounts were excreted in the urine of monkeys. When the drug is given perorally relatively small amounts are detected in the blood. This is largely due to the lack of absorption of streptomycin from the gastro-intestinal tract, as shown by the large amount of the drug recovered in the feces. Therapeutic blood concentrations can be maintained by repeated intramuscular injection. Following intravenous administration of streptomycin only 5 to 10 per cent of the dose can be demonstrated in the bile.—*Studies on the Absorption and Excretion of Streptomycin in Animals, R. B. Stebbins, O. E. Graessle & H. J. Robinson, Proc. Soc. Exper. Biol. & Med., October, 1945, 60: 68.—(F. B. Seibert)*

Cytotoxicity of Streptomycin and Streptothricin.—Several different preparations of streptomycin were tested on cultures of rabbits spleen and were found to have a uniformly low toxicity for wandering cells and fibroblasts. Streptothricin had a relatively low cytotoxicity for leucocytes and macrophages but showed a fairly high cytotoxicity for fibroblasts.—*Cytotoxicity of Streptomycin and Streptothricin, Dorothy H. Heilman, Proc. Soc. Exper. Biol. & Med., December, 1945, 60: 365.—(F. B. Seibert)*

Streptomycin in Body Fluids.—With the use of a streptomycin sensitive organism (*Staphylococcus aureus SM*), which was not inhibited by normal blood, by increase of pH of the medium, or by decrease in salt concentration, and a technique similar to that used for penicillin, it was possible to assay streptomycin in the blood, urine, tissue fluids of mice, rats, rabbits, dogs, monkeys and man.—*A Method for Determination of Streptomycin in Body Fluids, R. B. Stebbins & H. J. Robinson, Proc. Soc.*

Exper. Biol. & Med., June, 1945, 59: 255.—(F. B. Seibert)

p-Aminosalicylic Acid for Tuberculosis.—Data are given on the concentrations of various amino- and hydroxy-derivatives of benzoic acid which inhibit the growth of the tubercle bacillus *in vitro*. Of 60 compounds tested, 2-hydroxy-4-aminobenzoic acid (p-aminosalicylic acid, "PAS") was the most active, having bacteriostatic action in concentrations as low as $10^{-5.5}$ molal. Mice, rats and rabbits tolerated PAS well when fed 5 per cent of the compound in their food. Guinea pigs became emaciated, lost hair, and died in one to two weeks or less, nevertheless PAS had some protective action in animals injected intraperitoneally with 3 mg. of virulent human tubercle bacilli. PAS appeared to delay or arrest the progress of tuberculosis in some human cases. It was usually given by mouth in doses of 5, 3, 3 and 3 g. at four-hour intervals during the day. Owing to its rapid elimination from the body, the blood levels of PAS varied greatly, but usually averaged 3 to 6 mg. per 100 cc. of blood. Benzocaine and cycloform (the ethyl and isobutyl esters of p-aminobenzoic acid) were nearly as tuberculostatic *in vitro* as PAS, and might be useful in the local treatment of painful tuberculous lesions such as laryngeal tuberculosis. PAS has only a bacteriostatic action against the tubercle bacillus, but appears to be a useful drug.—*Chemotherapy of Tuberculosis: The Bacteriostatic Action of p-Aminosalicylic Acid (PAS) and Closely Related Substances upon the Tubercle Bacillus, together with Animal Experiments and Clinical Trials with PAS, J. Lehmann, Svenska läk.-tidning., August, 1946, 43: 2029.—(W. C. Tobie)*

p-Aminosalicylic Acid against Various Bacteria.—*Mycobacterium tuberculosis* (from sputum or from pure cultures) was grown in bouillon sealed on microscope slides by a method modified from that of Pryce (1941). The average concentration of p-aminosalicylic (PAS) which inhibited growth during eight days' incubation in four experiments was 0.153 mg. per 100 cc. of bouillon (10^{-5} molal), agreeing well with the results of Lehmann, see preceding abstract. PAS did not modify the tuberculin reaction in guinea pigs. PAS was tested against 19 species (a total of 37 strains) of non-acid-fast bacteria (pathogenic and nonpathogenic) but there was no bacteriostasis with concentrations less than 1.25 to 2.50 per cent of PAS, at which concentrations, 13 strains gave no growth. PAS did not protect mice injected with pneumococci or with tetanus toxin.—*Experimental Trials of p-Aminosalicylic Acid (PAS) against Various Kinds of Bacteria, O. Sievers, Svenska läk.-tidning., August, 1946, 43: 2041.—(W. C. Tobie)*

Clinical Experiences with p-Aminosalicylic Acid.—In treating tuberculosis, 4-aminosalicylic acid (p-aminosalicylic acid) (PAS) was usually given by mouth for three to four weeks, with one-week intervals without treatment. The daily dosage was 5, 3, 3 and 3 g., given at four-hour intervals. During treatment the average blood level was 5 mg. of PAS per 100 cc. of blood (extreme limits 1 to 10 mg.). In favorable cases, the fever usually gradually diminished over a period of days or weeks until the body temperature was normal. At the same time there was a reduction of the sedimentation rate, an increase in hemoglobin, a disappearance of tubercle bacilli from the sputum, an improvement in the roentgenological findings, and an increase in appetite. Relapses frequently occurred when treatment was suspended. The best results were obtained in exudative pulmonary tuberculosis, with 24 cases improved, 2 unimproved and 4 deaths. In productive fibrous cases, 9 improved, 7 did not improve and one died. Hilar tuberculosis (3 cases) and pleuritic tuberculosis (9 cases) showed improvement. Of 10 empyema pleurae cases (in some cases treated locally by injection of 5 to 10 per cent solutions of PAS without apparent benefit), 4 improved, 3 did not improve, and 3 died. However, extrapleural postoperative cavities

infected with *Mycobacterium tuberculosis* were treated very successfully by filling the whole cavity (usually of limited size) with a solution of PAS which was changed every second or third day. Healing resulted in 4 cases and improvement in 3 cases. Six cases of miliary tuberculosis or tuberculous meningitis terminated fatally despite treatment with PAS. PAS has a low toxicity. Toxemic symptoms (seen in a few cases) were: kidney irritation with a slight albuminuria, diffuse gastrointestinal discomfort and occasionally troublesome diarrhea.—*Clinical Experiences in the Treatment of Pulmonary Tuberculosis with PAS, G. Vallentin, Svenska läk.-tidning., August, 1946,* ***43:*** *2047.—(W. C. Tobie)*

Subtilin.—The antibacterial product, subtilin, obtained from *Bacillus subtilis* was found to be active chiefly against Gram-positive and also two Gram-negative bacteria, *Neisseria catarrhalis* and *N. gonorrhoeae*. Acid-fast organisms, including *Mycobacterium tuberculosis*, were also found to be susceptible to the antibiotic. The agent produced a bacteriostatic action in high dilution and a germicidal effect in greater concentration. A number of pathogenic higher fungi were also found to be susceptible to subtilin.—*Subtilin—An Antibiotic Produced by Bacillus subtilis: I. Action on Various Organisms, A. J. Salle & G. J. Jann, Proc. Soc. Exper. Biol. & Med., October, 1945,* ***60:*** *60.—(F. B. Seibert)*

Action of *Bacillus subtilis*.—Experiments demonstrate definite bacteriostatic and bacteriolytic properties of *Bacillus subtilis* and of "endosubtilysine" on cultures of tubercle bacilli. Subcutaneous administration of endosubtilysine in guinea pigs with experimental tuberculosis produced a retardation in the progression of lesions.—*Action du bacille subtil et de l'endo-subtilysine sur le bacille de Koch (souche d'Arloing et Courmont) et dans la tuberculose experimentale, H.-R. Olivier & L. de Saint-Rat, Rev. de la tuberc., 1946,* ***10:*** *50.—(V. Leites)*

Toxicity of Subtilin to Embryonic Tissue.—Subtilin, an antibiotic extracted from *Bacillus subtilis*, which was found to be antagonistic chiefly against Gram positive organisms, including *Mycobacterium tuberculosis* and other acid-fast bacteria, showed an extremely low toxicity for embryonic chick heart tissue fragments cultivated *in vitro*. Subtilin was found to be approximately twenty times more toxic to *Staphylococcus aureus* than to chick heart tissue, a remarkably low tissue toxicity. A unit of subtilin is defined as the amount contained in 1 cc. of the highest dilution capable of killing *Staphylococcus aureus* in ten minutes at 37° C. (F.D.A. phenol coefficient method.)—*Subtilin — Antibiotic Produced by Bacillus subtilis: II. Toxicity of Subtilin to Living Embryonic Tissue, A. J. Salle & G. J. Jann, Proc. Soc. Exper. Biol. & Med., January, 1946,* ***61:*** *23.—(F. B. Seibert)*

Tuberculosis in Swine.—Lesions classified as representative of tuberculosis in swine were examined by direct smears made from 102 submaxillary and mesenteric lymph nodes of swine, by culture and smears made from cultures with definite or suspicious growth, and by subcutaneous inoculation into chickens and guinea pigs. The chickens and guinea pigs did not react to avian and bovine tuberculin before inoculation. They were tested at about monthly intervals thereafter for about six months. Acid-fast bacilli resembling tubercle bacilli were found in smears from 95 (93.1 per cent) lesions. On culture, growth was obtained from 52 (50.9 per cent) of the lesions. The bacilli resembled tubercle bacilli, this being the only type encountered. From 15 (14.7 per cent) other lesions a few acid-fast bacilli were obtained on smears, but the number suggested that growth was either very slow or the organisms were carried over from the inoculum. As to the results of the inoculations into guinea pigs, only one swine specimen gave rise to generalized tuberculosis following guinea pig inoculation. Subinoculations in-

dicated that this was a bovine type of tubercle bacillus. Quite a number of guinea pigs reacted slightly to avian tuberculin at one test or another; and 65 (63.7 per cent) had caseous lesions at the site of injection, revealing in all but one smear acid-fast bacilli resembling tubercle bacilli. Of the inoculated birds, 5 did not live long enough; of the remaining 97 birds, 85 (87.6 per cent) became either positive to tuberculin or contained acid-fast bacilli in their tissues. Only in 2 reacting birds was it impossible to demonstrate acid-fast bacilli. Four of 7 swine specimens, negative on smears for acid-fast bacilli, were infective for chickens, thus making a total of 99 (97 per cent) of the specimens containing acid-fast bacilli resembling tubercle bacilli. Two chickens, inoculated with swine specimens negative on culture in chickens and guinea pigs, died before a definite conclusion could be reached. Of the remaining 100 specimens, 4 were chicken-negative but gave rise to local lesions with acid-fast bacilli, that is, 89 (89 per cent) of the specimens were positive for tubercle bacilli by chicken and/or guinea pig inoculation. In 88 per cent, the avian type was apparently found, and in 1 per cent, the bovine type. In 11 (11 per cent) of the swine specimens a definite demonstration of viable tubercle bacilli was not possible by culture or animal inoculation, but in smears, acid-fast bacilli were present in all but 3. It is reasonable to assume that, in some instances, bacilli were of such low virulence or so few in number that they could not produce lesions in animals.—*Tuberculous Lesions of Swine: II. Survey of Lesions Found in the Prairie Provinces, Especially in Alberta, J. C. Banker, Canad. J. Comp. Med., September, 1946, 10: 250.*—(*O. Pinner*)

Congo Red Test.—Interpretation of Congo red absorption tests falling between 40 and 90 per cent is equivocal. Bennhold declared that tests in this range indicate amyloid disease, the present work indicates the contrary. In a group of 73 patients tested at Sea View Hospital and found to be within the "doubtful" absorption range (30 to 89 per cent), retesting revealed markedly inconsistent results, suggesting the possibility of a purely fortuitous factor in determining the exact percentage of dye absorbed. The retesting of these patients indicated that in no way may the test be considered to have prognostic value; a patient showing 80 per cent absorption now may have 40 per cent absorption in three months. No correlation exists between the values shown in the Congo red tests and the nature of the primary disease. Justifiable conclusions from this research are: a test showing less than 90 per cent absorption cannot be considered diagnostic of amyloidosis; minimal or moderate amyloid disease may exist in the 40 to 90 per cent group and must be distinguished by other diagnostic measures.—*The Significance and Interpretation of the "Doubtful" Congo Red Test, I. J. Selikoff, Quart. Bull. Sea View Hosp., July, 1946, 8: 194.*—(*P. Q. Edwards*)

Congo Red Test.—A critical review and study of the existing methods of the Congo red test are given. The authors describe an improved technique. All patients in whom the Congo red in the plasma after four minutes was less then 15 mg. per thousand cc. were frankly amyloidotic. All those with 20 mg. or more, had no amyloidosis. As the distribution of the dye in the blood-stream is rapid and uniform, the four-minute test is generally sufficient. In liver disease the excretion of Congo red is slow. In nephrosis the excretion is rapid with presence of Congo red in the urine. In non-amyloidotic nephrosis, the disappearance of the Congo red is almost normal. There is not yet a satisfactory test for the recovery of Congo red in the urine.—*Tecnica fotometrica para la dosificacion del rojo congo del plasma sanguineo en el diagnostico de la amiloidosis, A. J. Soubrie & A. Patalano, An. Cáted. de pat. y clin. tuberc., June, 1945, 7: 143.*—(*W. Swienty*)

Anatomy of Right Upper Lobe Bronchus.—The right upper lobe bronchus has four divisions: the apical branch, the anterior branch, the posterior branch, and the axillary branch. Bronchoscopic and anatomical studies of the orifice of the right upper lobe bronchus led to the following classification: orifice with three openings, orifice with four openings, orifice with two openings. In the majority of cases (72.5 per cent) the right upper lobe bronchus was found to have three openings leading into the apical, anterior, and posterior branches. The axillary branch originates from the anterior or posterior division or from both and is directed towards the point of junction of the three lobes. An orifice with four openings was found only in 10 per cent of cases. In these the axillary branch originates directly from the lobar bronchus. It is visible in the inferior part of the bronchoscopic field between the openings of the anterior and posterior branch and opposite the apical branch. An orifice with two openings was present in 15.5 per cent of cases, in which the apical branch originated from a common trunk with the anterior or posterior branch. The common trunk is always situated in the upper part of the bronchoscopic field. The axillary branch originates from the anterior or posterior branch or from both.—*Orifices bronchoscopiques et anatomie bronchique du lobe supérieur droit, H. D'Hour, Y. Devin & P. Langevin, Rev. de la tuberc., 1946, 10: 81.*—(*V. Leites*)

The Segments of Lung.—It seems expedient to define a bronchopulmonary segment as that portion of lung served by a principal branch of a lobar bronchus, and thus the branch may be called a segmental bronchus. Such branches are relatively constant. The anatomy of bronchi may be studied by casts, dissections and bronchograms. The surface limits of a segment may be studied by distending it with air through its bronchus. Collapse or infection may be limited to a segment. Intersegmental boundaries are often marked by partial fissures on the surface of the normal lung. The radiological features of consolidation and collapse of each segment are described with a notation to the effect that, in nearly all so-called segmental consolidations, the radiographic shadow is smaller than would be expected from anatomical studies or inflation preparations. This is due to associated collapse. Proof of this lies in the presence of emphysema in adjoining segments, curving of interlobar fissures toward the shadow and compensatory displacement of the mediastinum or diaphragm. Conversely, there is nearly always some consolidation in a collapsed segment. There are nine major segments in the right lung and eight in the left lung. Levels of segmental boundaries are given with reference to a standard centering of the X-ray tube on the third costal cartilage at a distance of five feet. A segmental consolidation in the right middle lobe may be confused with interlobar effusion, but, in the lateral X-ray film, the fusiform homogeneous opacity of an effusion is absent. A bronchogram will usually distinguish between the two conditions by showing the relation of the middle lobe bronchus to the shadows. The most important application of segmental anatomy is the recognition of the fact that, where there is a segmental lesion, attention should be directed to the related bronchus where growth or stricture may be found. Thus, in bronchial carcinoma, an X-ray film revealing a large pulmonary opacity may be due not to growth but to consolidation or collapse due to growth in the affected bronchus. This will assist in estimating operability and in concentrating irradiation. In general, pneumonic lesions tend to transgress segmental boundaries or to affect less than a whole segment. This is especially true of atypical pneumonias. A truly segmental involvement is evidence against a simple pneumonic process. A knowledge of segmental anatomy with the direction of draining bronchi is important in planning postural drainage and in localization and external drainage of lung abscess. Acute putrid abscess is always segmental at its onset. Occasionally surgical resection

of separate bronchopulmonary segments is possible.—*The Segments of the Lung, A. F. Foster-Carter & C. Hoyle, Dis. of Chest, November–December, 1945, 11: 511.*—(*K. R. Boucot*)

Respiratory Air-flow.—The author has devised an instrument for measuring the instantaneous rate of air-flow during both phases of respiration. The instrument consists of a fine platinum wire suspended across the diameter of a tube. This wire is pivoted at one end and connected to a half-turn spring at the other. The wire is mounted in channels and, as air flows through the tube, the wire deflects in direct proportion to the rate of air-flow; this deflection is photographed on a moving film giving the air-flow curve or pneumotachogram. A portable instrument, operating upon a somewhat different principle, has been recently devised. Twenty-nine normal male subjects were tested with this portable instrument; similar curves were obtained in most of these patients. The curves obtained in bronchial asthma are diagnostic: the curves show marked damping especially in the expiratory phase; there is also a sharp return to zero at the end of expiration, a result produced in normal subjects by the application of external resistance. Among 8 patients with moderately advanced pulmonary tuberculosis, only 3 yielded abnormal curves; these 3 presented roentgen evidence of pulmonary fibrosis and diaphragmatic adhesions. First and early second-stage silicosis rarely produce any changes from the normal air-flow. Abnormal curves are usually seen in advanced second- and third-stage silicosis. The curves of 2 men with siderosis were within the normal range. Patients with chronic emphysema also manifested abnormal curves. Loss of pulmonary elasticity and internal resistance to air-flow are the chief factors in producing abnormal changes in air-flow curves. This technique appears to be of value in differentiating between lung deposits which cause fibrosis and those which produce X-ray changes without fibrosis.—*Respiratory Air Flow Characteristics and Their Relation to Certain Lung Conditions Occurring in Industry, L. Silverman, J. Indust. Hyg. & Toxicol., September, 1946, 28: 183.*—(*H. R. Nayer*)

New Method of Spirometry.—In order to avoid the disadvantages of the usual methods of bronchospirometry a new procedure is proposed based on compression of one hemithorax, thus permitting the evaluation of the function of each lung separately. A detailed description of the method is given, which combines thoracography, spirometry and hemithoracic compression. In cases where the patient does not tolerate complete blockage of the hemithorax, the method permits to vary the degree of compression. The institution of compression produces immediately a compensatory increase in the function of the contralateral lung, as evidenced by an increase of the amplitude of respirations. Even in cases of reduced or absent motility of one hemithorax due to pleural symphysis, fibrothorax or unexpandable lung, compression of the other side provokes a compensatory increase of the functional capacity—a fact which would not have become evident with the method of simultaneous bilateral bronchospirometry. The imperfections of the new method reside mainly in its technical difficulties, but it presents no discomfort for the patient.—*Une nouvelle methode d'examen fonctionel séparé des poumons, P. Labesse, Rev. de la tuberc., 1946, 10: 153.*—(*V. Leites*)

Respiratory Centre.—The neurons which constitute the respiratory centre are diffusely distributed through the reticular formation of the caudal half of the bulb. Those in the ventral reticular substance overlying the inferior olivary nuclei are concerned with inspiration. Those in the dorsal reticular substance are concerned with expiration. Excitatory connections between the constituent neurons of a given centre provide for coördination of contraction of widely distributed respiratory muscles. Inhibitory connections between the two centres provide for

alternation of contraction of inspiratory and expiratory muscles. The neurons of the respiratory centre are sensitive to the chemical and physical constitution of their fluid environment. They are excited by impulses impinging upon them by way of collaterals of the major sensory and motor tracts. Under combined chemical and synaptic stimulation these neurons discharge impulses repetitively. The more intense the chemical stimuli and the more numerous the synaptic stimuli, the greater is the frequency of discharge of impulses and the greater is the number of neurons active. Since the inspiratory neurons have the lowest threshold, inpiration is the dominant phase of respiration. In the absence of inhibitory influences which act upon the inspiratory centre from without, its discharge is continuous, and maintained tonic inspirations or apneusis results. The depth of the apneusis is a function of the sum of all the stimuli acting upon the neurons of the inspiratory centre. Two inhibitory mechanisms operate to interrupt rhythmically this repetitive activity of the inspiratory centre, namely the vagal inhibitory mechanism and the pneumotaxic mechanism. As the lungs inflate during inspiration, impulses which originate in pulmonary stretch receptors and which travel centrally over afferent fibers of the vagus nerves excite the expiratory centre. The inspiratory centre is reciprocally inhibited and expiration results. As the lungs deflate, the afferent inflow diminishes, the inspiratory centre escapes and the cycle repeats. The pneumotaxic mechanism plays an analogous although a subsidiary rôle under most circumstances. Thus impulses originating in the inspiratory centre are transmitted rostrally to the pneumotaxic centre, and are then relayed caudally to the expiratory centre. Excitation of the expiratory centre leads to reciprocal inhibition of the inspiratory centre, whereupon the circuit ceases to function and the cycle repeats. When the vagus nerves are cut the pneumotaxic centre maintains the rhythm of breathing. The rhythm of breathing is impressed upon the respiratory centre by inhibitory mechanisms operating from without, it is not an expression of properties inherent in the neurons of the centre. The depth of breathing is determined by the sensitivity of these neurons to their environment and by the excitatory influences exerted by the many afferents which impinge upon them. The rate of breathing is determined by the excitability of the neurons of the centre and by the activity of the inhibitory mechanisms which rhythmically interrupt their repetitive discharge. (Author's summary.)—*Organization of the Respiratory Center, R. F. Pitts, Physiol. Rev., October, 1946, 26: 609.*—(*G. C. Leiner*)

Pulmonary Ventilation and Anoxemia.—The relation of pulmonary ventilation to arterial oxygenation has been studied in 9 resting subjects breathing 10.5 per cent oxygen. Arterial oxygen saturation was found to be extremely sensitive to even small changes in pulmonary ventilation. Increasing the resting ventilation by half, a change of which the subject is scarcely aware, may increase arterial saturation by 10 to 20 per cent. Doubling or tripling the resting ventilation produces smaller increases in saturation. Arterial oxygen tension bears a direct and linear relation to respiratory minute volume. With increasing ventilation the alveolar and arterial oxygen tensions increased 1.5 and 2.0 times as fast as the corresponding carbon dioxide tensions fell. Considerable improvement in oxygenation may be obtained, without symptoms of acapnia, by a small increase in resting ventilation. This effect is due to improved mixing of tracheal and alveolar air, replacement of carbon dioxide by oxygen and temporary elevation of the respiratory quotient, while subjects are in an unsteady state. The influence of changes in pulmonary ventilation must be evaluated in any clinical application of anoxia. (Author's summary.)—*The Effect of Pulmonary Ventilation on Anoxemia, C. S. Houston, Am. J. Physiol., July, 1946, 146: 613.*—(*G. C. Leiner*)

Pulmonary Volume Receptors.—The responses of single afferent fibers of the vagus nerve to changes in lung volume were recorded in cats under Dial anesthesia. The fibers could be divided into distinct groups according to the rate at which their end-organs adapted to lung inflation. The slowly adapting receptors had on the average a lower inflation threshold than the rapidly adapting receptors. Some receptors of both kinds responded to forced deflation of the lungs, but none were found which responded only to deflation. The conduction velocity, measured in 20 fibers and corrected to body temperature, was between 8 and 44 meters per second. The differences in adaptation and threshold of the two afferent fiber groups when compared with the two reflexes evoked by inflation of the lungs, suggest that impulses from slowly adapting endings inhibit inspiration, while those from the rapid adaptors excite inspiration. This evaluation of function adequately accounts for the respiratory responses to both inflation and deflation of the lungs. Only the slowly adapting fibers are in action in eupnea. A change in their threshold contributes to the increased rate of respiration in hyperpnea. The principal function of the rapidly adapting fibers appears to be a reinforcement of depth of certain deep inspirations which have been initiated through other mechanisms. (Authors' summary.)—*A Unitary Analysis of Pulmonary Volume Receptors, G. C. Knowlton & M. G. Larrabee, Am. J. Physiol., September, 1946,* ***147:*** *100.*—*(G. C. Leiner)*

Alveolar Gas Pressure.—The limitations inherent in direct methods for sampling alveolar air become critical during experiments on exercising subjects. An indirect method for calculating alveolar gas pressures has been devised which requires simply the determination of arterial pCO_2 and pO_2 and pCO_2 of inspired and expired air. A theoretical analysis indicates that alveolar gas pressures so determined represent the physiologically effective mean pressures and are not subject to errors introduced by "time" and "space" factors. By this indirect method the effective alveolar pressures may be determined during exercise without encountering the difficulties inherent in the classical direct methods. (Authors' summary.)—*On the Determination of the Physiologically Effective Pressures of Oxygen and Carbon Dioxide in Alveolar Air, R. L. Riley, J. L. Lilienthal, Jr., D. D. Proemmel & R. E. Franke, Am. J. Physiol., September, 1946,* ***147:*** *191.*—*(G. C. Leiner)*

Alveolar and Arterial Oxygen Pressure.—By means of new techniques, measurements have been made in man of the oxygen pressure gradient existing between the alveolar air and the peripheral arterial blood, during rest and exercise, at sea level and at simulated altitude. At rest the gradient averaged 9 mm. Hg and during exercise 16.5 mm. Hg; the development of anoxia produced no significant changes in the size of the gradients. A method is presented for differentiating the total alveolar arterial oxygen pressure gradient into its two main components: membrane resistance and venous admixture. A theoretical analysis of the experimental data indicates that when the level of oxygenation was high (sea level) the observed pressure gradient resulted for the most part from the admixture of venous blood entering from poorly ventilated alveoli, the bronchial circulation, the Thebesian and anterior cardiac veins and perhaps other sources. By contrast, a low level oxygenation (anoxic anoxia) the gradient resulted largely from the pressure head which must develop across the pulmonary membrane to effect the transfer of the required volume of oxygen. Exercise, by exerting a physiological stress on the mechanisms serving the transfer of oxygen from alveolar air to arterial blood, evokes an integrated series of respiratory and cardiovascular adaptations, one of which is the increase of the alveolar-arterial oxygen pressure gradient. The diffusion constant of the lung, calculated from the experimental observations, averaged 21 at rest (range 12 to 36) and increased during exercise to an

average of 62 (range 50 to 76). (Authors' summary.)—*An Experimental Analysis in Man of the Oxygen Pressure Gradient from Alveolar Air to Arterial Blood during Rest and Exercise at Sea Level and at Altitude, J. L. Lilienthal, Jr., R. L. Riley, D. D. Proemmel & R. E. Franke, Am. J. Physiol., September, 1946, 147: 199.*—(*G. C. Leiner*)

Hemodynamics in Phosgene Poisoning.—Circulatory measurements were made on anesthetized and unanesthetized dogs before and after exposure to phosgene. There was a decrease of the pulse rate and of the arterial pressure. The venous pressure remained unchanged. The blood volume decreased. There was a prolonged pulmonary circulation time, an increased arterial-venous oxygen difference and hemoconcentration. Spasms of vessels were seen. Death is due primarily to an interference with oxygen uptake because of pulmonary edema. If the acute stage of pulmonary edema with its attendant anoxic anoxia is survived, circulatory failure may become a more important factor in the ultimate outcome. Venesection is not indicated in phosgene poisoning.—*Hemodynamics in Pulmonary Irritant Poisoning, H. M. Patt, J. M. Tobias, M. N. Swift, S. Postel & R. W. Gerard, Am. J. Physiol., October, 1946, 147: 329.*—(*G. C. Leiner*)

Respiration in Cardiac Dyspnea.—Records of chest movements during breathing were made with a Marey pneumograph in normal subjects, in patients with cardiac dyspnea and in patients with bronchial asthma. The relation of expiratory to inspiratory phase in 5 normal persons at rest varied from 1.30:1 to 1.96:1, the average being 1.61:1. After exercise the average fell to 1.39:1. In 11 patients with heart disease at rest the ratio was between 1.52:1 and 2.90:1, with an average of 2.17:1. After exercise the ratio remained unchanged or decreased only slightly. In 2 patients with bronchial asthma the ratio was at rest 2.14 in average; after exercise it was 1.81. The intravenous administration of aminophyllin produced in cardiac and in bronchial asthma a marked relative shortening of the expiratory phase. The vital capacity increased in patients with cardiac and in patients with bronchial asthma after injection of aminophyllin; in a normal subject there was only an insignificant increase. From these observations it is concluded that bronchospasm is present in both groups of patients.—*Abnormalities of the Respiratory Pattern in Patients with Cardiac Dyspnea, H. E. Heyer, Am. Heart J., October, 1946, 32: 457.*—(*G. C. Leiner*)

Pulmonary Permeability. — Pulmonary permeability was studied by injecting ethyl alcohol intravenously and determining the output in the expired air. A detailed description of the method and its causes of error is given. Clinical experiments did not permit to establish any correlation between the condition of the lung and the elimination of alcohol. Disturbances of pulmonary permeability are not considered to be clinically significant. What counts in pulmonary permeability, is not so much the total surface of the lung but the relation between surface and alveolar volume. Most lung diseases did not modify this relation with the exception of generalized emphysema in which a slight reduction of the factor was found.—*Est-il possible de faire l'examen fonctionel du poumon par des tests d'élimination des substances volatiles? Y a-t-il des troubles cliniques de la perméabilité pulmonaire?, R. Monod & M. Cara, Le Poumon, September-October, 1945, 1: 257.*—(*V. Leites*)

Phrenic Nerve Response to Lung Inflation.—The influence of changes in lung volume on the discharge of impulses by single phrenic motor neurones was investigated in cats anesthetized with Dial or decebrated under ether. Inflation of the lungs inhibits the phrenic discharge for a length of time that increases with the volume of inflation. Large inflations, in addition to this well-known inhibition of inspiration, have an excitant action which is revealed by a brief burst of motor impulses as the lungs expand. This

excitant action is of short duration and has a threshold which is relatively independent of the rate of distention. The inspiration-exciting reflex is independent of vascular pressure receptors since it is not reduced during occlusion of the pulmonary artery. Both the inhibition and excitation of inspiration by lung inflation are eliminated by cutting both vagus nerves. It is suggested that these reflexes are due to impulses from two distinct sets of pulmonary receptors of different thresholds and different rates of adaptation. Increased activity of phrenic motor neurones is caused by artificial distention of the lungs only when the lungs are inflated to a volume which exceeds that reached in eupneic respiration. Following a suddenly induced pneumothorax the next inspiration is very much prolonged, but the frequency of discharge follows exactly the same time course as in normal inspiration up to the time when the normal discharge is abruptly curtailed. Inspiration is thus uninfluenced by impulses from pulmonary stretch receptors until its termination is suddenly brought about by the inspiration-inhibiting reflex. The inspiration-exciting reflex initiated by superinflation of the lungs is inactive in normal eupneic breathing, but may serve to increase the depth of any unusually deep inspiration. (Authors' summary.)—*Excitation and Inhibition of Phrenic Motoneurones by Inflation of the Lungs, M. G. Larrabee & G. C. Knowlton, Am. J. Physiol., September, 1946, **147:** 90.*—(*G. C. Leiner*)

Pulmonary Calcifications.—It was recently reported that pulmonary calcification is more frequently associated with sensitivity to histoplasmin than to tuberculin or coccidiodin in the central eastern portion of the United States. In the past few years pulmonary calcification in tuberculin-negative persons has been demonstrated in many of the central eastern states and other agents than the tubercle bacillus have been considered as the possible causative factor. Aronson presented evidence favoring coccidioidomycosis among the Indians in the Southwest. Smith has shown that the area of prevalence of tuberculin negative pulmonary calcification corresponds with the endemic area of histoplasmosis. The study presented is based on data by Palmer and additional material collected with the help of the National Tuberculosis Association, the United States Public Health Service and a large number of schools of nursing. There is a wide variation of pulmonary calcification geographically but the positive reaction to histoplasmin closely parallels the incidence of calcification but no such parallel with positive tuberculin reactions was apparent in this material. Four times as many nurses showed calcification in Kansas City as in Philadelphia and over four times as many were sensitive to histoplasmin. All but 38 of 532 tuberculin-negative persons were histoplasmin positive. There are no generally accepted criteria for the recognition of pulmonary calcification. A total of 6,199 student nurses were X-rayed and tested with tuberculin and histoplasmin. Pulmonary calcification was found in 698, 57 of whom were tuberculin-positive, 109 tuberculin- and histoplasmin-positive. Little association could be found between questionable calcification and sensitivity to either antigen. An attempt was made to differentiate between calcification presumably due to tuberculosis and that presumably due to histoplasmosis or an immunologically similar disease. The total number of calcific deposits did not seem to differ in the two groups and no striking or characteristic feature of the individual lesions has been noted which would be of value in determining the etiological factor. It is possible that histoplasmosis is responsible for most instances of scattered multiple bilateral calcifications. Massive calcification in the hilar zones has frequently been considered pathognomonic of healed tuberculosis, but from these studies it is evident that such areas need not represent previous tuberculous infection. It is likely that sensitivity to histoplasmin is an indication of previous infection with the fungus *Histoplasma capsulatum* or an immunologically related organism, and that infection with these organisms is

not necessarily serious or fatal but is widespread in subclinical form. Since it is impossible to distinguish between the calcifications resulting from the two infections it may be inferred that the roentgenographic appearance of the benign form of histoplasmosis may resemble tuberculosis during the active phase. The tuberculin reaction and finding of tubercle bacilli are, therefore, essential in the diagnosis of pulmonary tuberculosis.—*Pulmonary Calcification: Roentgenographic Observations in Relation to Histoplasmin and Tuberculin Reactions, H. B. Zwerling & C. E. Palmer, Radiology, July, 1946, 47: 59.*—(*G. F. Mitchell*)

Pulmonary Calcifications and Age.—Intrathoracic calcifications following tuberculous infection were studied in 1,457 American Indian children who were the control group in previously reported studies on BCG vaccination. In this group, 198 developed tuberculosis; calcifications subsequently occurred in 49 of these children. Calcified foci were seen most frequently during the second and third years following the first lesion. In the youngest age group, calcification developed in 76.5 per cent of tuberculosis cases. The rate declined in older children, and was found in only 2.4 per cent of tuberculosis cases in which the disease started between the ages of 15 and 19. It is concluded that the frequency with which calcification develops in pulmonary tuberculosis varies directly with the age at which the tuberculous lesions begin.—*Variation with Age in the Frequency of Tuberculous Pulmonary Calcification, R. H. High & H. B. Zwerling, Pub. Health Rep., December 6, 1946, 61: 1769.*—(*M. Pinner*)

Pulmonary Calcifications.—A total of 113 cases of disseminated pulmonary calcifications were studied. Of these, 64 cases were derived from an X-ray survey of 15,980 school children in Kansas City. An additional 49 cases came from other sources. Of the total 113 cases, 69, or 61.1 per cent, had multiple bilateral calcifications, while 44 had calcifications of the miliary type. Calcifications in the hilar lymph nodes were seen in 73.5 per cent of the former type and in 50.0 per cent of the latter type. The prevalence of calcifications increased with age, being zero in children under 4 years and increasing to 10 per thousand in the age group of 16 to 18. They were about four times as frequent in whites as in Negroes. No persons with calcifications reacted to tuberculin only; 93.5 per cent of persons with disseminated calcifications reacted to histoplasmin alone. Two cases reacted both to tuberculin and to histoplasmin, and 2 reacted to neither substance. In the whole group of 113 cases, 96.3 per cent reacted to histoplasmin, and only 4 did not react to it. None of the group reacted only to tuberculin. This study provides further circumstantial evidence in support of the working hypothesis that, at least in some parts of the U.S.A., disseminated calcifications are caused by whatever agent produces sensitivity to histoplasmin.—*Disseminated Pulmonary Calcification, R. H. High, H. B. Zwerling & M. L. Furcolow, Pub. Health Rep., January 3, 1947, 62: 20.*—(*M. Pinner*)

Calcifications in Spleen.—As incidental findings in an X-ray survey in Kansas City, 20 cases of splenic calcifications were found. It is in this geographical area of the U.S.A. that about 40 per cent of white school children (and a somewhat lower percentage of Negro school children) react to histoplasmin. Among children with pulmonary calcifications, about 80 per cent react to histoplasmin, and it was found the 78.6 per cent of children with splenic calcifications reacted to histoplasmin. The close similarity in the prevalence of histoplasmin reactors among children with pulmonary and with splenic calcifications suggests that extrapulmonary calcifications are probably caused by the same agent that causes pulmonary calcifications, and that produces skin sensitivity to histoplasmin. Splenic calcifications indicate that the causative agent may be disseminated hematogenously, and apparently without

causing obvious clinical disease. It is not likely that tuberculosis is the most frequent cause of splenic calcifications in this geographical region.—*Calcifications in the Spleen, R H. High, Pub. Health Rep., December 6, 1946, 61: 1782.*—(*M. Pinner*)

Upper Respiratory Infections.—The factors involved in the control of upper respiratory infections, mostly caused by hemolytic streptococci, are discussed. For climatic reasons, training camps should be situated in the southern part of the country. Overcrowding must be avoided: minimum living space requirements, established usually according to the standards of the Medical Department, are at times far from ideal in regard to epidemic disease control. In some epidemic areas, even these minimal standards have not been adhered to. The introduction, at frequent intervals, of new and usually susceptible recruits into battalions where streptococcal infection is prevalent, is an ideal condition for building up and maintaining epidemics. Instead, recruits should be grouped as fast as possible into self-contained batallions into which no new recruits should be transferred during the training period. After a batallion has been moved out, the vacated living quarters should be thoroughly cleaned, aired and disinfected. Present barracks designs foster the spread of epidemics. The incidence of streptococcal infections is more closely related to the number of men housed under one roof than to the space allotted per man. A maximum of 4 men per room is an ideal practical arrangement. Mere partitioning of barracks into cubicles, with a common aisle between them, is ineffective. Changes in basic design can be considered for future construction only. Multitudes of organisms, including hemolytic streptococci, are harbored in floor dust and bedding lint and become air-borne whenever the dust is disturbed or the bed clothes are shaken. Elimination of dry sweeping and oiling of bedding and floor, with the exception of rough concrete and linoleum, are highly recommended. Oil on bed clothes is hardly noticeable; on floors, it soaks in within a few hours; it immobilizes dust and bacteria but has no bactericidal effect unless a germicide has been added. Ultraviolet irradiation of air and surfaces has been effective in killing bacteria but is expensive, it would be extremely useful in communicable disease wards of hospitals. Ultraviolet light plus oiling reduces air-borne organisms by as much as 80 per cent, thus diminishing cross-infection. Propylene and triethylene glycol used in aerosols condense on, and kill bacteria in the air, but do not kill organisms present on surfaces. Their wide-spread use has not yet proved practicable, but they may be found useful in selected places. Patients with upper respiratory infections should be segregated in hospital wards limited to the exclusive care of such cases. Ideally, these wards should offer single rooms and be managed as contagious disease wards, with no free communication allowed between patients and the medical and nursing staff employing the proper isolation technique. If this should not be possible, each bed should be placed in an improvised cubicle made of sheets or built-in partitions. After recovery the patient should be moved to a ward of similar design, reserved for patients convalescing from upper respiratory infections. If, at this stage, cultures from nose and throat are positive for hemolytic streptococci, penicillin should be administered. After two consecutive cultures have been negative, the patient can be returned to his company. This process should not require more than fourteen days and will save many man-days. In order to correct some of the factors which at present operate to produce cross-infections and new cases, the following measures are recommended: (*1*) segregation, in the waiting rooms, of patients with upper respiratory infections; (*2*) assigning of staggered sick-call hours for the various activities or units of the command; (*3*) proper sterilization of all items of common use. Mass prophylaxis with sulfonamides is not satisfactory; infections caused by drug resistant strains become epidemic. Other agents now available are not encourag-

ing. The results of active immunization with killed whole organisms of prevalent types, as well as type-specific antigenic components of of the cells do not justify the use of this method for mass protection. Passive immunization with gamma globulin has, on the whole, been disappointing.—*Control of Upper Respiratory Diseases, (not signed), Bull. U. S. Army Med. Dept., December, 1946, 6: 675.—(O. Pinner)*

Upper Respiratory Infections.—In 13 patients who were hospitalized because of upper respiratory infection electrocardiographic changes were observed. Group A beta-hemolytic streptococci were present in the nasopharyngeal cultures of 5 of these patients; in one case a Group B and in one a Group G beta-hemolytic streptococcus was found. In 6 cases repeated cultures showed only the usual nasopharyngeal organisms. The most frequent electrocardiographic changes were T-wave inversions and depressions. With the exception of one, none of the patients was acutely ill. None of these patients had cardiac signs or symptoms when the electrocardiographic changes were observed. Only one patient developed clinical manifestations suggesting rheumatic fever. All patients recovered. A nonspecific toxic etiology is assumed. Sulfonamide administration may be an additive factor.—*Electrocardiographic Changes Occurring during Upper Respiratory Infections, D. Young, Am. Heart J., September, 1946, 32: 383.—(G. C. Leiner)*

Cure of Early Bronchiectasis by Pneumothorax.—This report from the Mt. Sinai Hospital, New York, based on 6 cases, gives an enthusiastic recommendation for the treatment of bronchiectasis by pneumothorax in all cases where the usual measures fail (chemotherapy, bronchoscopic aspirations, partial drainage, etc.) and where absence of adhesions permit a selective collapse of the diseased lobe. The collapse need be maintained for only six months, as complete recovery was attained in this period, and the reëxpanded lobes have remained cured over periods ranging from seven to thirteen years. Favorable results with pneumothorax therapy in chronic cases is apparently possible if a selective collapse can be obtained; the failures may be treated by radical surgical measures. Pneumothorax therapy, if employed judiciously, has no mortality, no morbidity and no serious complications. In the pathogenesis of bronchiectasis, bronchial dilatation is caused by the weakening of the muscular and elastic tissues by infection. This dilatation is augmented by cough, by bronchial obstruction, by the pull on the injured bronchi exerted during inspiration by the expanding thorax transmitted through atelectatic lung instead of through elastic parenchyma; and by the pull of the more negative intrapleural pressure which is found in the presence of atelectasis or fibrosis. With these factors in mind the objectives in treatment of bronchiectasis are: (*1*) to diminish the trauma of respiration by decreasing its force; (*2*) to minimize the trauma of cough; (*3*) to counteract the increased pull on the diseased bronchi because of lowered intrapleural pressures; (*4*) to eliminate the infection in lung and bronchi. Case reports show that pneumothorax can accomplish these objectives, since cough promptly decreased and then disappeared within one to four weeks; infection subsided promptly and permanently. In case reports it is also shown that suppurative bronchopneumonia can result in the rapid development of saccular bronchiectasis, and that pneumothorax therapy is not only an effective measure in preventing the development of bronchiectasis, but also in its treatment. In these cases, pneumothorax was tried only after two months of treatment by other measures: postural drainage, repeated bronchoscopic aspirations, generous use of expectorants (iodides), frequent inhalations of 5 per cent carbon dioxide in oxygen, inhalations of steam as well as chemotherapy. When these measures failed, pneumothorax was tried and brought about cures. These cases were treated in the prepenicillin period, and the best way to use this drug, whether by inhalation spray, by catheter instillation, by injection or whether by all methods simultaneously, has not yet been determined.—*Pneumothorax Therapy for*

Early Bronchiectasis, H. Hennell, J. Thoracic Surg., August, 1946, 15: 239.—(W. M. G. Jones)

Flagellate in Bronchiectasis.—It is generally recognized that only one flagellate normally lives in the mouth of man—genus *Trichomonas Donne*. A patient is reported in whose sputum many flagellates were discovered. He was admitted to hospital with diagnosis of malaria. Despite vigorous antimalarial therapy pyrexia continued. He started to expectorate a considerable amount of foul smelling sputum. A lung abscess was diagnosed two weeks after admission and under sulphonamide therapy pyrexia declined, but quantity of sputum and respiratory distress were unchanged. Original examination of the sputum revealed numerous flagellates and spirochaetes in addition to the customary fauna. Further examinations, with careful attention to avoidance of contamination, revealed the same picture. Gradually they decreased and finally no flagellates were found. Patient died three weeks after admission. Necropsy showed bilateral pulmonary fibrosis, with numerous irregularly shaped cavities filled with foul smelling gelatinous secretion. Histological examination confirmed the diagnosis of long-standing cylindrical bronchiectasis. No flagellates were noted in cavity secretions post-mortem. The flagellate recovered from the sputum was pear-shaped, 10 to 15 μ in length, with two equal flagella at the narrow end. It is considered that this was either *Trichomonas tenax* or the flagellate stage of *Dimastigamoeba gruberi*, which had entered the lungs via the mouth and had there found in the mucopurulent material of the bronchiectatic cavities an ideal medium on which it had rapidly multiplied. The authors state that there is little reason to doubt that these flagellates are nothing more than commensals living saprophytically on the decaying contents in the bronchiectatic cavity and have apparently no pathological significance.—*Occurrence of a Flagellate in the Sputum of a Case of Bronchiectasis, G. O. Lehmann & J. T. Prendiville, Brit. M. J., February 2, 1946, 1: 158.*—(D. H. Cohen)

Bronchiectasis and Atelectasis.—Four cases of atelectasis with bronchiectasis are described. In all 4 cases, reaeration and reëxpansion of the collapsed portion of the lung occurred. In 3, there was retrogression of the bronchiectasis; in the other case, it persisted.—*Bronchiectasis and Atelectasis: Temporary and Permanent Changes, F P. L. Lander, Thorax, September, 1946, 1: 198.*—(A. G. Cohen)

Atelectatic Bronchiectasis.—Shadows representing the right middle lobe in the conventional postero-anterior view are often difficult to interpret. Lateral views help but sometimes the shadow of the atelectatic lobe is hardly denser than that of the normal lung. In fact, this is often also true in postero-anterior views. It is believed that the lordotic position brings the interlobar septum more or less in the plane of the rays. Fourteen cases of atelectatic bronchiectasis of the right middle lobe were seen in an eighteen-month period. In most cases cough, with or without purulent sputum, had been present since an early age. Slight variable physical signs referable to the middle lobe were found in less than 50 per cent of cases. Roentgenograms in the conventional views were normal in many cases. Abnormal findings included increased striation at the right base, hazy opacity in the region of the lower pole of the right hilum extending a little way into the lung field and enlargement of the lower pole of the root of the right lung with or without downward retraction. The lordotic view showed a triangular shadow with its base on the right cardiac border below the hilum and its apex somewhere in the middle of the lower lung field. The upper and lower margins were sharp. This area was denser than the surrounding lung field. The shadow was seldom homogeneous. Bronchograms taken in the lordotic position served to dissociate the shadows of the middle lobe bronchi from those of the lower lobe. Lipiodol was seen to enter the involved area and bronchiectases were found.—*Atelectatic Bronchiectasis of the Right*

Middle Lobe, A. T. Doig, Tubercle, November, 1946, 27: 173.—(A. G. Cohen)

Hemoptysis and Bronchoscopy.—Among 332 bronchoscopies performed for various reasons, 80 (24 per cent) were indicated by a previous hemoptysis. Hemorrhagic tracheobronchitis was found in 38 cases, 19 of these had active or arrested pulmonary tuberculosis. Bronchoscopy revealed bronchogenic carcinoma in 12.5 per cent of the cases, benign bronchial tumors in 5 per cent, bronchiectasis in 15 per cent, bronchial stenosis in 7.5 per cent. There was no demonstrable bronchial abnormality in 7.5 per cent of cases. It is concluded that in 90 per cent of the cases with hemoptysis bronchoscopy permitted to determine the tracheobronchial origin and the specific cause of the bleeding.—*Hemoptysie et endoscopie bronchique, une statistique de 80 cas, P. Mounier-Kuhn, C. Ollagnier & A. Persillos, Le Poumon, July-August, 1946, 2: 13.—(V. Leites)*

Aerosol in Asthma.—Eighty-four patients with acute or chronic asthmatic dyspnea were treated with inhalation of nebulized bronchodilator drugs, chiefly 0.2 per cent Aleudrine (isopropyl adrenaline) which is reported to have ten times the bronchodilator power of adrenalin, with or without 0.5 to 1.0 per cent Adrianol (neosynephrin). The results are classified according to the age, sex, duration of treatment, type of dyspnea and intensity of the symptoms, and by dominant etiological factor. All 84 patients are stated to have experienced some relief, most of them markedly so.—*La therapeutique pro longee des dyspnoes asthmatiformes par les aerosols medicamenteux pneumodilatateurs, R. Charlier, Rev. belge sc. méd., February, 1946, 16: 42.—(E. Bogen)*

Treatment of Pulmonary Actinomycosis.—This report deals with 19 cases from Barnes Hospital since 1925. (Total number in all reports, therefore, 52). Of these 19 cases, all had proved laboratory diagnosis, but the author believes many cases may have been missed due to the difficulty in finding the organism, which in some cases took as long as two to three years. Eight of the 19 cases have died as a result of the infection or its complications; 6 of these died by generalization of the disease. In 5 patients the disease first made its appearance in the intestinal tract, and these all did poorly. Only 6 professed any association with cattle. Detailed reports of the cases that recovered are given. Surgical drainage or resection of diseased tissue was performed in all patients, supplemented by penicillin or sulfonamides, or small doses of X-ray irradiation in certain instances. Other adjuvants used formerly were thymol and potassium iodide in addition to surgery. The author was struck by the remarkable benefits of penicillin to control the most severe symptoms, and believes this drug will supercede all others, to supplement the surgical treatment. About 25 per cent of all infections are present in lung and chest wall; 60 per cent in the jaw and cervical regions. The pulmonary picture presented by these cases is that of a chronic pneumonitis accompanied by fever, cough and especially chest pain. The infection extends over many months. Lung abscesses tend to be small and multiple. The disease may extend in any direction, peripherally involving pleura, periosteum and ribs. Chest wall sinuses drain for a long time. Medial extension may enter mediastinum or spinal canal. Entrance into blood stream proves rapidly fatal. Diagnosis is best made from tissue section. Material directly aspirated from an abscess or obtained through a bronchoscope is more satisfactory than discharge from a sinus tract. The fungi can be cultured and this should always be done in suspected cases.—*Treatment of Pulmonary Actinomycosis with a Report of Seven Arrested Cases, J. K. Poppe, J. Thoracic Surg., April, 1946, 15: 118.—(W. M. G. Jones)*

INDEX OF ABSTRACTS

Abbott, O. A., and de Oliveria, H. R. Pneumothorax following bronchoscopy, 116
Abbott, W. E. See Hirshfeld, J. W., *et al.*, 118
Abel, H. A., and Katz, H. L. Tuberculous meningitis, 45
Abercromby, B. M. L. Foreign bodies, 115
Abnormal X-ray findings in surveys, 133
Abscess, Lung, 77
—, mediastinal, Anterior, 124
—, Tuberculous, following penicillin, 155
Absorption and excretion of streptomycin, 168
—, Pleural, of dyes, 68
Accelerated sensitization, 163
Acevedo, R. C., Giuntini, L. S., and Croxatto, O. C. Pulmonary adenomatosis, 108
Acid-fast bacilli, False, 158
— microörganisms, Atypical, 56
— organisms in gastric contents, 43
Acids, Fatty, and bacterial growth, 157
Actinomycosis, Pulmonary, 83
—, —, Treatment of, 182
Active tuberculosis, Negative tuberculin reaction in, 23
Acute empyema, Penicillin in, 119
— silicosis, 102
Adams, A. J., Captain, and Fitzpatrick, L. J., Major. Nerve block in treatment of thoracic injuries, 126
Adams, R. Primary lung tumors, 107
Adenoma of bronchus, 109
Adenomatosis, Pulmonary, 108
Adults, Primary tuberculosis in, 16, 134, 135
Aerosol in asthma, 182
Age, Pulmonary calcifications and, 178
Aged, Pneumonia in, 72
Agenesis of lung, 68
Air-borne tubercle bacilli, 61
Air-flow, Respiratory, 173
Aksel, A. Mediastinal tumors removed by surgery, 123
Albino rat, Endemic pneumonia in, 73
Alexander, A. E., and Soltya, N. A. Surface-active substances and tubercle bacilli, 159
Alexander, H. Tuberculous bronchitis, 25
Alibert, A. See Biedermann, A., *et al.*, 132
Alkylresorcinols on tubercle bacilli, 59
Allen, A. W., Linton, R. L., and Donaldson, G. A. Pulmonary embolism, 95
Allen, Margaret F. See Furcolow, M. L., *et al.*, 7
Allen, W. See Hampton, S. F., *et al.*, 107
Allergy, Spread of dye in, 65
—, Tuberculin, 66
Alley, F. H. Extrapleural pneumothorax, 144
Alveolar and arterial oxygen pressure, 175
— cell tumor, 107
— gas pressure, 175
Anal fistula, 130
Anaphylactic reactions to Congo red, 130
Anatomy of right upper lobe bronchus, 172
Anderson, R. G., and Chapman, E. M. Physical diagnosis, 69
Andrejew, A. Respiration of tubercle bacilli, 156
Anemia, Pernicious, and tuberculosis, 139
Aneurysm of innominate artery, 128
Anoxemia, Pulmonary ventilation and, 174
Antagonistic growth substances, 164
Anterior mediastinal abscess, 124
Antibiotic from *Ramalina reticulata*, 165
Antibodies against pneumococci, 71
— to pneumococci and polysaccharides, 70
Antituberculosis work, Controversial points in, 131
Arnold, M. Indications for collapse therapy, 140
Aronovitch, M. Atelectasis, 92
—, —., and Vineberg, A. M. Anterior mediastinal abscess, 124
Aronson, J. D., and Palmer, C. E. BCG vaccination in Indians, 63
Arterial, Alveolar and, oxygen pressure, 175
Artery, innominate, Aneurysm of, 128
Arthritis, Tuberculous, 153
Artificial pneumothorax, 31, 32
Ashton, N. Tuberculomata of liver, 152
Aspiration, Bronchial, in atelectasis, 93
Aspirations, bronchoscopic, Tubercle bacilli in, 159
Asthma, 105, 107
—, Aerosol in, 182
— in Southwest Pacific, 105
—, Premenstrual, 107
—, Surgery for, 106
Atelectasis, 14, 92
— after hemoptysis, 28
—, Bronchial aspiration in, 93
—, Bronchiectasis and, 181
Atelectatic bronchiectasis, 181
Atresia, Esophageal, and tracheo-esophageal fistula, 124

Atypical acid-fast microörganisms, 56
—— pneumonia, Primary, 74
Aufses, Beatrice H. Instrument tray for chest surgery, 146
Averous, M. Extrapleural pneumothorax and penicillin, 145
Avian tuberculosis in man, 29
Aycock, T. B. See Brantigan, O. C., *et al.*, 37
Azparren, J. S., and Juarrero, V. P. Toxic granulations in tuberculosis, 44

Bacilli, acid-fast, False, 158
——, Bovine type, 158
——, Chemical inhibition of, 165
——, Hansen's, and cultured leprosy bacilli, Disparity between, 157
——, leprosy, cultured, Hansen's bacilli and, Disparity between, 157
——, tubercle, Air-borne, 61
——, ——, Alkylresorcinols on, 59
——, ——, and urea, 59
——, ——, Culture of, 160
——, ——, Demonstration of, in sputum, 43
——, ——, Diagnostic culture of, 160
——, ——, Dissociation of, 158
——, ——, Growth of, in liquid media, 54
——, ——, —— rate of, 56
——, ——, —— stimulation of, 160
——, ——, in bronchoscopic aspirations, 159
——, ——, —— gastric contents, 43
——, ——, —— tuberculous lesions, 140
——, ——, Metabolism of, 57, 156
——, ——, Preservation of, 159
——, ——, Respiration of, 156
——, ——, Streptomycin resistant strains of, 168
——, ——, Submerged growth of, 159
——, ——, Surface-active substances and, 159
——, ——, Virulence of, 57
——, ——, Vole, 161
Bacillus subtilis. Action of, 170
Bacillus, tubercle, Culture medium for, 55
Bacteria and fungi, Fatty material in, 157
——, various, P-aminosalicylic acid against, 169
Bacterial growth, Fatty acids and, 157
Bacteriostasis *in vitro*, 164
Baker, H., and Wilson, D. A. Experimental pulmonary collapse, 92
Ballooned, inert and residual cavities, 141
Banker, J. C. Tuberculosis in swine, 170
Bariéty and Barrabé. Tuberculosis of serous membranes, 49
Bariéty, M., and Paillas, J. Perforation of tension cavity, 136
——, ——., —— ——, ——. Tubercle bacilli in bronchoscopic aspirations, 159
——, ——., —— ——, ——. X-ray appearance of draining bronchi, 27
Baron, J. See Dufourt, A., *et al.*, 137
Barrabé and Bariéty. Tuberculosis of serous membranes, 49
Barrett, M. R., and Thomas, D. Hydatid cyst, 85
Bass, H. E. Inoculation tuberculosis, 150
Bassin, S. See Bobrowitz, I. D., *et al.*, 112
BCG, 62, 63
—— in Denmark, 62
——, Local reactions to, 163
—— vaccination, 61, 163
—— —— in Indians, 63
Bed-rest, 29
Beds for tuberculosis, 3
Benatt, A. J. Pneumoperitoneum against hemoptysis, 36
——, ——. ——., and Berg, W. F. Electrocardiogram in pneumoperitoneum, 36
Bence, A., Peroncini, J., and Rey, R. C. Biopsy of lung cancer, 109
Bence, A. E. Bronchial aspiration in atelectasis, 93
Berblinger, W. Genito-urinary tuberculosis, 155
——, ——. Tuberculosis of uterus, 154
Berczeller, A., and Frank, Grace. False acid-fast bacilli, 158
Berg, S. Intrapleural and extrapleural pneumothorax, 33
Berg, W. F., and Benatt, A. J. Electrocardiogram in pneumoperitoneum, 36
Bergquist, S., and Ernberg, T. Primary tuberculosis in adults, 16
Bernard, E., and Weil, J. Duration of pneumothorax, 142
Bernstein, A. See Klosk, E., *et al.*, 100
Bernstein, I. J., and Selikoff, I. J. Anaphylactic reaction to Congo red, 130
——, ——. ——. See Fox, T. T., *et al.*, 29
Bertheau, M. Empyema in pneumothorax, 142
Beryllium poisoning, 104
Besançon, F., and Delarue, J. Tuberculous necrosis, 162
Biedermann, A., Alibert, A., Mery, J., Lerolle, M., and Paillas, P. Fluoroscopy in railroad employees, 132
Biggs, A. D. Primary tuberculosis, 15

Bilateral pleural effusions, 47
—— pneumothorax, Pneumonolysis in, 33
Billingslea, T. H., and Smyth, C. J. Lung abscess, 77
Bindslev, J., and Jensen, K. A. Virulence of tubercle bacilli, 57
Biological changes in *M. ranae*, 60
Biopsy of lung cancer, 109
Birkhaug, K. Immunology of tuberculosis, 64
——, ——., and Böe, J. Spread of dye in allergy, 65
——, ——., —— Schjelderup, H. Hematology in experimental tuberculosis, 66
Blahd, M., Leslie, M. I., and Rosenthal, S. R. Infectiousness of closed cases, 12
Blahd, Margery. See Rosenthal, S. R., *et al.*, 163
Blajat Pena, I. Pulmonary segments, 88
Bloch, H. Metabolism of tubercle bacilli, 156
Blood coagulation in pleural cavity, 117
—— prothrombin in tuberculosis, 25
—— sedimentation, Heparin and, 44
Bobrowitz, I. D., Edlin, J. S., Bassin, S., and Woolley, J. S. Penicillin in bronchiectasis, 112
Body fluids, Streptomycin in, 168
Böe, J., and Birkhaug, K. Spread of dye in allergy, 65
Boeck's sarcoidosis, 86
Bone tuberculosis, 152
—— ——, Surgery in, 153
Bornstein, P. K. Streptomycin in tuberculous meningitis, 167
Boulenger, P., and Maclouf, A.-C. Accelerated sensitization, 163
——, ——. See Courcoux, A., *et al.*, 163
Bouquet, F. See Perdigon, E., *et al.*, 164
Bovard, P. G., and Ritter, W. L. Hazards in fire-brick industry, 104
Bovine type bacilli, 158
Bradbury, F. C. S. Avian tuberculosis in man, 29
Braillon, J. Extrapleural pneumonolysis, 145
Brannon, E. S. See Merrill, A. J., *et al.*, 126
Brantigan, O. C. Open pneumonolysis, 143
——, ——. ——., Aycock, T. B., Hoffman, R., and Welch, H. J. Relaxing thoracoplasty, 37
Brean, H. P., and Kane, L. W. Pulmonary tuberculosis in medical students, 132
Breath sounds, 43
Brewer, L. A. Plombage, 36
Brewer, L. A., III, Major. See Buford, T. H., Major, *et al.*, 129
Brjum, V. I. Atelectasis, 14
Brock, R. C. New incision in thoracoplasty, 147
Bronchi, Draining, 26
——, ——, X-ray appearance of, 27
Bronchial aspiration in atelectasis, 93
—— disease, Penicillin in, 112
Bronchiectasis, 114
—— and atelectasis, 181
—— —— dextrocardia, 113
——, Atelectatic, 181
——, Bronchoscopy in, 114
——, early, Cure of, by pneumothorax, 180
——, Flagellate in, 181
——, Nondisabling, 110
——, Penicillin in, 112
——, Surgery for, 114
——, Treatment of, 111
——, X-ray diagnosis in, 113
Bronchitis, Tuberculous, 25
Bronchoscopic aspirations, Tubercle bacilli in, 159
—— treatment, 139
Bronchoscopy, Hemoptysis and, 182
—— in bronchiectasis, 114
——, Pneumothorax following, 116
Bronchospirometry, Postpneumothorax, 34
Bronchus, Adenoma of, 109
——, Cancer of, 108
——, right upper lobe, Anatomy of, 172
Brown, C. L. See Durant. T. M., *et al.*, 73
Brown, H. A., Hinshaw, H. C. Streptomycin toxicity, 42
Bruce, T. Postpneumothorax bronchospirometry, 34
Brunner, A. Displacement of mediastinum, 122
Buford, T. H., Major, and Burbank, B., Major. Traumatic wet lung, 98
——, ——. ——., ——, Samson, P. C., Major, Brewer, L. A., III, Major, and Burbank, B., Major. Recovery from hemolytic staphylococcus aureus bacteremia, 129
Buggs, C. W. See Hirshfeld, J. W., *et al.*, 118
Buie, L. A., and Jackman, R. J. Anal fistula, 130
Bullous emphysema, 100
Bunn, P. A., McDermott, W., Hadley, Susan J., and Carter, Anne C. Pneumonia, 71
Burbank, B., Major, and Buford, T. H., Major. Traumatic wet lung, 98
——, ——., ——. See Buford, T. H., Major, *et al.*, 129

Burdon, K. L. Disparity between Hansen's bacilli and cultured leprosy bacilli, 158
——, ——. ——. Fatty material in bacteria and fungi, 157
Burgos, R. See Gomes, F. D., *et al.*, 31
Burns, Pulmonary complications in, 94
——, Pulmonary lesions in, 94
Buu-Hoi and Jouin, J. P. Chemical inhibition of bacilli, 165

Calafat, A. M. Tuberculosis of nasal septum, 51
Calcifications in spleen, 178
——, Pleural, 49
——, Pulmonary, 177, 178
——, ——, and age, 178
Cameron, G. M., and Castles, R. Demonstration of tubercle bacilli in sputum, 43
Camiel, M. R. Draining bronchi, 26
——, ——. ——. Tuberculous arthritis, 153
Canabal, E. J., and Piaggio Blanco, R. A. Insufflated hydatid cyst, 28
Cancer, lung, Biopsy of, 109
—— of bronchus, 108
Canetti, G. Tubercle bacilli in tuberculous lesions, 140
Cantonnet Blanch, H. See Cantonnet Blanch, P., *et al.*, 63
Cantonnet Blanch, P., Cantonnet Blanch, H., and Lieutier, H. BCG, 63
Cara, M., and Monod, R. Pulmonary permeability, 176
Carcopino, C., and Joly, H. Results of thoracoplasty, 146
Cardiac dyspnea, Respiration in, 176
Cardiospasm in tuberculosis, 139
Carmodi, M. G. See Van Ordstrand, H. S., *et al.*, 104
Carryer, H. M., Prickman, L. E., Maytum, C. K., and Koelsche, G. A. Asthma, 105
Carter, Anne C. See Bunn, P. A., *et al.*, 71
Cases, closed, Infectiousness of, 12
Cassinelli, J. F., and Lasnier, E. P. Hydatid cyst, 84
Castles, R., and Cameron, G. M. Demonstration of tubercle bacilli in sputum, 43
Cat, Tuberculosis from a, 45
Cattle tuberculosis in U.S.A., 67
Cavity, pleural, Blood coagulation in, 117
——, tension, Perforation of, 136
Cell tumor, Alveolar, 107
Cellular transfer of tuberculin reactivity, 162
Centre, Respiratory, 173
Cervia, T., and Gutierez, V. Hepatitis and tuberculosis, 28
Cervical lymph nodes, Tuberculous, 51
Changes, Biological, in *M. ranae*, 60
Chapman, C. B., and Whorton, C. M. Miliary tuberculosis, 44
Chapman, E. M., and Anderson, R. G. Physical diagnosis, 69
Charlier, R. Aerosol in asthma, 182
Chase, M. W. Cellular transfer of tuberculin reactivity, 163
Chauvet, M. Cardiospasm in tuberculosis, 139
Chemical inhibition of bacilli, 165
Chemistry of chronic lesions, 139
Chemotherapy, Experimental, 40
Chest examinations in discharged soldiers, 132
—— films, Miniature, 6
—— operations, Support for patients during, 146
——, Pain in, 124
—— surgery, Instrument tray for, 146
—— wounds, 126
—— ——, penetrating, Circulation in, 126
Children, Disseminated tuberculosis in, 15
——, Tuberculosis in, 12, 13, 14
Chronic epituberculosis, 136
—— lesions, Chemistry of, 139
—— pulmonary infections, Penicillin in, 77
Chute, R. Genito-urinary tuberculosis, 154
Circulation in penetrating chest wounds, 126
Cities, major, Tuberculosis in, 3
——, Tuberculosis mortality in, 3
Clark, D., and Gilmore, J. H. Coccidioidin tests, 82
Classification of tuberculosis of skin, 151
Claviculectomy, Partial, 148
Climie, H. Tuberculin patch tests, 7
Clinical experiences with p-aminosalicylic acid, 169
Closed cases, Infectiousness of, 12
Coagulation, Blood, in pleural cavity, 117
Coccidioidin tests, 82
Cochrane, A. L. Tuberculosis among prisoners of war, 10
Cohen, B. See Halbert, P., *et al.*, 69
Cohen, R. Tuberculosis and pregnancy, 22
Cohen, R. C. Lower-lobe tuberculosis, 24
——, ——. ——. Pneumoperitoneum, 35
Cohen, S. See Kinsman, J. M., *et al.*, 72
Cohn, M. L., and Corper, H. J. Diasone treatment, 40
Collapse, Lobar and segmental, of lung, 89
—— of right middle lobe, 90
——, pulmonary, Experimental, 92
——, ——, Postoperative, 93
——, Segmental, of lung, 90
—— therapy, Indications for, 140

Complications of pneumothorax, 143
—— —— thoracoplasty, 146
——, Pulmonary, in burns, 94
Comstock, G. W. See Kramer, M., *et al.*, 5
Congenital tuberculosis, 149
Congo red, Anaphylactic reactions to, 130
—— —— test, 171
Conjunctiva, Primary tuberculosis of, 151
Control division, Tuberculosis, 2
——, tuberculosis, in Minneapolis, Principles of, 131
Cook, E., Greene, L., and Hinshaw, H. Streptomycin in urinary tract tuberculosis, 53
Cor pulmonale, 127
Corper, H. J. Immunology of tuberculosis, 65
——, ——. ——., and Cohn, M. L. Diasone treatment, 40
——, ——. ——., —— Stoner, R. E. Culture of tubercle bacilli, 160
——, ——. ——., —— ——, ——. ——. Diagnostic culture of tubercle bacilli, 160
Corre, L. Local reactions to BCG, 163
Cory, R. A. S. Oxidized gauze in thoracoplasties, 148
Courcoux, A., Boulenger, P., and Maclouf, A.-C. BCG vaccination, 163
Couve, Ph. Tuberculosis among prisoners of war, 11
Croxatto, O. C. See Acevedo, R. C., *et al.*, 108
Croxatto, R. See Ortuzar, R., *et al.*, 81
Cruzat, M. See Ortuzar, R., *et al.*, 81
Culture, Diagnostic, of tubercle bacilli, 160
—— medium for tubercle bacillus, 55
—— of tubercle bacilli, 160
Cultured "leprosy bacilli," Disparity between Hansen's bacilli and, 157
Culver, G. J. Pulmonary Hodgkin's disease, 110
Cummins, S. L. Tubercle bacilli and urea, 59
Cure of early bronchiectasis by pneumothorax, 150
Cyst, Hydatid, 84, 85
——, ——, Insufflated, 28
—— of diaphragm, 121
Cystic disease, 100
Cytotoxicity of streptomycin and streptothricin, 168

D'Alonso, C. A. See Kinsman, J. M., *et al.*, 72
Daniels, W. B. See Kinsman, J. M., *et al.*, 72
Danzer, J. T. X-ray peculiarities in mental deficiency, 21
D'Arcy Hart, P., and Garland, T. O. Tuberculosis control in Newfoundland, 131
Dathe, R. A., and Nathan, D. A. Pericarditis following upper respiratory infection, 128
Davidson, C. S., and Levenson, S. M. Pulmonary complications in burns, 94
——, ——. ——. See Finland, M., *et al.*, 94
Davis, B. D., and Dubos, R. J. Growth of tubercle bacilli in liquid media, 54
Daza, F. X-ray diagnosis in bronchiectasis, 113
De Abreu, M. Photoroentgenography in tuberculosis prophylaxis, 4
Deficiency, mental, X-ray peculiarities in, 21
Deflections, Ventricular, in tuberberculosis, 29
Delarue, J., and Besançon, F. Tuberculous necrosis, 162
Delaunay, A., Vendrely, R., and Pages, J. Chemistry of chronic lesions, 139
Dell, D., and Jerram. Tuberculin testing, 7
Delord, M. Epituberculosis, 136
DeNardi, J. M. See Van Ordstrand, H. S., *et al.*, 104
Denize, A. See Hochberg, L. A., *et al.*, 147
Denmark, BCG in, 62
——, Tuberculosis in, 1
de Oliveria, H. R., and Abbott, O. A. Pneumothorax following bronchoscopy, 116
de Paula, A. Tuberculosis in prostitutes, 133
de Saint-Rat, L., and Olivier, H. R. Action of *Bacillus subtilis*, 170
Deshmukh, P. L. Primary thoracoplasty, 146
Devin, Y. See D'Hour, H., *et al.*, 172
Dextrocardia, Bronchiectasis and, 113
D'Hour, H., Devin, Y., and Langevin, P. Anatomy of right upper lobe bronchus, 172
Diacoumopoulos, A. See Lemoine, J. M., *et al.*, 32
Diagnosis, Physical, 69
——, X-ray, in bronchiectasis, 113
Diagnostic culture of tubercle bacilli, 160
Diaphragm, Cyst of, 121
Diasone treatment, 40
Dick, W. P. Symptoms in pulmonary tuberculosis, 138
Discharged soldiers, Chest examinations in, 132
Disease, bronchial, Penicillin in, 112
——, Cystic, 100
——, Heart, 2
——, Hodgkin's, Pulmonary, 110
——, Hydatid, of pleura, 120
——, pulmonary, Penicillin inhalation in, 75
Diseases, Fungus, 81
Displacement of mediastinum, 122

Disseminated tuberculosis in children, 15
Dissociation of tubercle bacilli, 158
Distribution, Global, of tuberculosis, 2
Doig, A. T. Atelectatic bronchiectasis, 181
Donaldson, G. A. See Allen, A. W., *et al.*, 95
Dormer, B. A., Friedlander, J., and Wiles, F. J. Syphilis of lung, 80
Drainage, Intracavitary, 149
Draining bronchi, 26
—— ——, X-ray appearance of, 27
Drea, W. F. Alkylresorcinols on tubercle bacilli, 59
Dry pleurisy, 118
Dublin, L. I. Tuberculosis Association program, 131
Dubos, R. J. Fatty acids and bacterial growth, 157
——, ——. ——., and Davis, B. D. Growth of tubercle bacilli in liquid media, 54
Dufourt, A. Primary tuberculosis in adults, 135
——, ——., and Mounier-Kuhn, P. Chronic epituberculosis, 136
——, ——., Mounier-Kuhn, P., and Baron, J. Pulmonary and endobronchial tuberculosis, 137
Dumarest, F., and Mollard, H. Complications of pneumothorax, 143
Duncan, J. T. Fungus diseases, 81
Durant, T. M., Sokalchuk, A. J., Norris, C. M., and Brown, C. L. Influenza pneumonia, 73
Duration of pneumothorax, 142
Dust particle size, 103
Duszynski, Diana Olga, and Scatchard, G. N. Miniature chest films, 6
Dutrey, J., and Vaccarezza, R. F. Genetic factor in tuberculosis, 17
Dye, Spread of, in allergy, 65
Dyes, Pleural absorption of, 68
Dyspnea, cardiac, Respiration in, 176

Early bronchiectasis, Cure of, by pneumothorax, 180
Ebrill, D., and Elek, S. D. Tuberculous abscess following penicillin, 155
Echinococcus of pleura, 121
Edlin, J. S. See Bobrowitz, I. D., *et al.*, 112
Edwards, P. W., and Logan, J. Pneumoperitoneum, 35
Effusions, Pleural, 46
——, ——, Bilateral, 47
——, ——, Prognosis in, 47
——, ——, Radiology of, 47
Ehrenpreis, B., and McDonald, J. B. Primary atypical pneumonia, 74
Eichwald, M., and Singletary, W. V. Loeffler's syndrome, 78
Electrocardiogram in pneumonia, 73
—— —— pneumoperitoneum, 36
Elek, S. D., and Ebrill, D. Tuberculous abscess following penicillin, 155
Embolism, Pulmonary, 95
——, ——, Treatment of, 94
Embryonic tissue, Toxicity of subtilin to, 170
Emmart, W. T. See Smith, M. I., *et al.*, 167
Emphysema, 99
——, Bullous, 100
——, Mediastinal, 122
——, Surgical, 116
Employees, railroad, Fluoroscopy in, 132
Empyema, acute, Penicillin in, 119
—— in pneumothorax, 142
——, Nontuberculous, 118
——, Postpneumonic, 119
——, Putrid, 120
——, tuberculous, Treatment of, 48
Endemic pneumonia in albino rat, 73
Endobronchial tuberculosis, Pulmonary and, 137
Endotoxoid, Tubercle, 38
Eosinophilia, Tropical, 79, 80
Epidemic pleurodynia, 118
Epituberculosis, 136
——, Chronic, 136
Epstein, N. N. Tuberculosis of skin, 150
Erikson, J. Streptococcus pneumonia, 76
Ernberg, T., and Bergquist, S. Primary tuberculosis in adults, 16
Erythema nodosum, 128
Esophageal atresia and tracheo-esophageal fistula, 124
Even, R., and Lecoeur, J. Ballooned, inert and residual cavities, 141
——, ——., —— ——, ——. Fever after refills, 142
Examinations, Chest, in discharged soldiers, 132
Excretion of streptomycin, Absorption and, 168
Experiences, Clinical, with p-aminosalicylic acid, 169
Experimental chemotherapy, 40
—— Loeffler's syndrome, 80
—— pulmonary collapse, 92
—— tuberculosis, Hematology in, 66
Extrapleural pneumonolysis, 145
—— pneumothorax, 144, 145
—— —— and penicillin, 145
—— ——, Intrapleural and, 33

Exudative tuberculosis, Pathophysiology of, 22

False acid-fast bacilli, 158
Fatti, L., Florey, M. E., Joules, H., Humphrey, J. H., and Sakula, J. Penicillin in acute empyema, 119
Fatty acids and bacterial growth, 157
—— material in bacteria and fungi, 157
Feeney, N. Pulmonary embolism, 95
Feinstone, W. H. Tuberculostatic substances, 165
Feldman, D. J., and Lewis, H. P. Pleural effusions, 46
Feldman, W. H. See Hinshaw, H. C., *et al.*, 166
Fernandes, H. P. Bilateral pleural effusions, 47
Fever after refills, 142
——, Premenstrual, in tuberculosis, 137
Films, chest, Miniature, 6
Findings, X-ray, Abnormal, in surveys, 133
Fine, A., and Steinhausen, T. B. Nondisabling bronchiectasis, 110
Fink, I. See Hochberg, L. A., *et al.*, 147
Finland, M., Ritvo, M., Davidson, C. S., and Levenson, S. M. Pulmonary lesions in burns, 94
Finlayson, Margaret K. Growth stimulation of tubercle bacilli, 160
Finnish sanatoria, 6
Fire-brick industry, Hazards in, 103
Fistula, Anal, 130
——, tracheo-esophageal, Esophageal fistula and, 124
Fitzpatrick, L. J., Major, and Adams, A. J., Captain. Nerve block in treatment of thoracic injuries, 126
Flagellate in bronchiectasis, 181
Flippin, H. E. See Zintel, H. A., *et al.*, 40
Florey, M. E. See Fatti, L., *et al.*, 119
Floyer, M. A., and May, H. B. Penicillin in bronchiectasis, 112
Fluids, body, Streptomycin in, 168
Fluoroscopic screens, 134
Fluoroscopy in railroad employees, 132
Foci, Round, 136
Foley, G. E. Submerged growth of tubercle bacilli, 159
Foreign bodies, 115
Fourestier, M. Photofluorographic survey, 133
Fox, T. T., Sokol, D. D., and Bernstein, I. J. Ventricular deflections in tuberculosis, 29
Frank, Grace, and Berczeller, A. False acid-fast bacilli, 158
Franke, R. E. See Lilienthal, J. L., *et al.*, 175
——, ——. ——. See Riley, R. L., *et al.*, 175
Freedlander, B. L. Experimental chemotherapy, 40
Friedlander, J. See Dormer, B. A., *et al.*, 80
Friedmann, I. Culture medium for tubercle bacillus, 55
Frisk, A. R. Bacteriostasis *in vitro*, 164
Fungi, Fatty material in bacteria and, 157
Fungus diseases, 81
Furcolow, M. L., High, H. H., and Allen, Margaret F. Histoplasmin reactors, 7
——, ——. ——. See High, R. H., *et al.*, 178

Gaisford, W. F. Tuberculosis in children, 14
Garland, T. O., and D'Arcy Hart, P. Tuberculosis in Newfoundland, 131
Gas pressure, Alveolar, 175
Gastric contents, Acid-fast organisms in, 43
—— ——, Tubercle bacilli in, 43
Gauze, Oxidized, in thoracoplasties, 148
Geiger, W. B., Green, S. R., and Waksman, S. A. Inactivation of streptomycin, 168
Geiser, P., Schaub, K., and Staub, H. Penicillin inhalation in pneumonia, 72
Generator, X-ray, 6
Genetic factor in tuberculosis, 17
Genito-urinary tuberculosis, 154, 155
Gerard, R. W. See Patt, H. M., *et al.*, 176
Gilmore, J. H., and Clark, D. Coccidioidin tests, 82
Gines, A. R., and Rebull Richieri, V. BCG, 62
Ginsburg, G. S. Primary tuberculosis in adults, 135
Giuntini, L. S. See Acevedo, R. C., *et al.*, 108
Global distribution of tuberculosis, 2
Glover, R. E. Preservation of tubercle bacilli, 159
——, ——. —— Tuberculosis in hamsters, 161
Godard, F. See Perdigon, E., *et al.*, 164
Gold, E. M., and Wright, D. O. Loeffler's syndrome, 78
Goldfarb, A. A., and Seltzer, I. Primary tuberculosis of conjunctiva, 151
Gomez, F. D., Negro, J. C., and Burgos, R. Phrenic nerve paralysis, 31
Gotta, G., and Noguera, O. F. Pleural calcifications, 49
Gould, D. M. Nontuberculous lesions in mass X-ray surveys, 69
——, ——. ——. Tuberculosis survey, 4

Graessle, O. E. See Stebbins, R. B., *et al.*, 168
Grandstaff, E. H. Postoperative pulmonary collapse, 93
Granulations, Toxic, in tuberculosis, 44
Granules of *M. tuberculosis*, 58
Graubner, E. Workmen's compensation for silicosis, 103
Green, S. R. See Geiger, W. B., *et al.*, 168
Greene, L. See Cook, E., *et al.*, 53
Grenville-Mathers, R. Premenstrual fever in tuberculosis, 137
Grez, A. Bronchoscopy in bronchiectasis, 114
Gross, R. E., and Scott, H. W., Jr. Esophageal atresia and tracheo-esophageal fistula, 124
Grove, P. R. Tuberculous osteoarthritis, 52
Growth, bacterial, Fatty acids and, 157
—— of tubercle bacilli in liquid media, 54
—— rate of tubercle bacilli, 56
—— stimulation of tubercle bacilli, 160
——, Submerged, of tubercle bacilli, 159
—— substances, Antagonistic, 164
Gutierez, V., and Cervia, T. Hepatitis and tuberculosis, 28
Gutman, S. Vole tubercle bacilli, 161

Habeeb, W. J. Silicosis, 102
Hadley, Susan J. See Bunn, P. A., *et al.*, 71
Halbert, P., Cohen, B., and Perkins, M. E. Pneumococcal hemolysin, 69
Hale, C. H., and Robbins, L. L. Collapse of right middle lobe, 90
——, ——. ——., —— ——, ——. ——. Lobar and segmental collapse of lung, 89
——, ——. ——., —— ——, ——. ——. Segmental collapse of lung, 90
Hall, S., and Pagel, W. Congenital tuberculosis, 149
Hamman, L. Mediastinal emphysema, 122
Hampton, S. F., Wine, M. B., Allen, W., Thompson, G. S., and Starr, M. P. Asthma, 107
Hamsters, Tuberculosis in, 161
Handler, N. J., and Spain, D. M. Cor pulmonale, 127
Hansen's bacilli and cultured "leprosy bacilli," Disparity between, 157
Hardy, Janet. B. Persistence of tuberculin sensitivity, 162
Harrell, G. T. See Travis, O. T., *et al.*, 67
Harrington, F. E. See Myers, J. A., *et al.*, 12
Harrison, T. R. Pain in chest, 124
Harvey, Elinor B., and Hogg, P. Pulmonary thrombosis, 97
Hatt, F. Welding hazards, 104
Hawkins, S. F., and Thomas, G. O. Lower-lobe tuberculosis, 24
Hazards in fire-brick industry, 103
——, Welding, 104
Head, J. R. Intracavitary drainage, 149
Healy, M. J., and Katz, H. L. Nontuberculous empyema, 118
Heart disease, 2
Heidelberger, M., MacLeod, C. M., Kaiser, S. J., and Robinson, B. Antibodies to pneumococci and polysaccharides, 70
Heilman, Dorothy H. Cytotoxicity of streptomycin and streptothricin, 168
Heilman, F. R., Hinshaw, H. C., Nichols, D. R., and Herrell, W. E. Pharmacology of streptomycin, 41
Heller, R. J. Hemoptysis, 27
Hematology in experimental tuberculosis, 66
Hemodynamics in phosgene poisoning, 176
Hemolysin, Pneumococcal, 69
Hemolytic staphylococcus aureus bacteremia, Recovery from, 129
Hemoptysis, 27
—— and bronchoscopy, 182
——, Atelectasis after, 28
——, Pneumoperitoneum against, 36
Hennell, H. Cure of early bronchiectasis by pneumothorax, 180
Heparin and blood sedimentation, 44
Hepatitis and tuberculosis, 28
Herbut, P. A. Alveolar cell tumor, 107
——, ——. ——., and Kinsey, F. R. Experimental Loeffler's syndrome, 80
Herman, M., Levin, Florence C., and Thompson, S. E. Primary tuberculosis, 17
Herrell, W. E. See Heilman, F. R., *et al.*, 41
Heyer, H. E. Respiration in cardiac dyspnea, 176
Hiatt, J. S., Jr., and Martin, D. S. Moniliasis, 82
High, H. H. See Furcolow, M. L., *et al.*, 7
High, R. H. Calcifications in spleen, 178
——, ——. ——., and Zwerling, H. B. Pulmonary calcifications and age, 178
——, ——. ——., Zwerling, H. B., and Furcolow, M. L. Pulmonary calcifications, 178
Hilleboe, H. E. Abnormal X-ray findings in surveys, 133
——, ——. ——. Controversial points in antituberculosis work, 131
——, ——. ——. Minimal tuberculosis, 19
Hilton, G. Cancer of bronchus, 108

Hinshaw, H. C., and Brown, H. A. Streptomycin toxicity, 42
——, ——. ——., Pfuetze, K. H., and Feldman, W. H. Streptomycin in tuberculosis, 166
——, ——. ——. See Cook, E., *et al.*, 53
——, ——. ——. See Heilman, F. R., *et al.*, 41
Hirshfeld, J. W., Buggs, C. W., Abbott, W. E., and Pilling, M. A. Nontuberculous empyema, 118
Histoplasmin reactors, 7
History of penicillin, 164
Hochberg, L. A., Fink, I., and Denize, A. Revision thoracoplasty, 147
Hodes, P. J., and Wood, F. C. Tropical eosinophilia, 79
Hodgkin's disease, Pulmonary, 110
Hoffman, R. See Brantigan, O. C., *et al.*, 37
Hogg, P., and Harvey, Elinor B. Pulmonary thrombosis, 97
Hollingworth, R. K. Intrathoracic sympathetic nerve tumors, 123
Holm, J. BCG in Denmark, 62
Holmes, J. Tuberculosis in Denmark, 1
Holt, Rebecca A., and Lynch, J. P. Pulmonary actinomycosis, 83
Honkanen, A. Finnish sanatoria, 6
Houston, C. S. Pulmonary ventilation and anoxemia, 174
Hughes, R. See Van Ordstrand, H. S., *et al.*, 104
Humphrey, J. H., and Joules, H. Penicillin inhalation in pulmonary disease, 75
——, ——. ——. See Fatti, L., *et al.*, 119
Hydatid cyst, 84, 85
—— ——, Insufflated, 28
—— disease of pleura, 120

Ignatowskaja, N. N., and Rabich-Shchebro, V. A. Phrenic nerve paralysis, 30
Illing, R. B. Tuberculosis in children, 13
Immunology of tuberculosis, 64, 65
Inactivation of streptomycin, 168
Incipient pulmonary tuberculosis, 19
Incision, New, in thoracoplasty, 147
Indians, BCG vaccination in, 63
Indications for collapse therapy, 140
Industrial plant, Resurvey of, 5
Industry, fire-brick, Hazards in, 103
Inert, Ballooned, and residual cavities, 141
Infarcts, Pulmonary, 96
Infection, Primary, 135
——, respiratory, upper, Pericarditis following, 128
Infections, pulmonary, chronic, Penicillin in, 77
——, respiratory, Upper, 179, 180
——, Typhoid and salmonella, 87
Infectiousness of closed cases, 12
Inflation, lung, Phrenic nerve response to, 176
Influenza pneumonia, 73
Inhalation, Penicillin, in pneumonia, 72
——, ——, in pulmonary disease, 75
Inhibition, Chemical, of bacilli, 165
Injuries, thoracic, treatment of, Nerve block in, 126
Innominate artery, Aneurysm of, 128
Inoculation tuberculosis, 150
Institutions, mental, Tuberculosis in, 6
Instrument tray for chest surgery, 146
Insufflated hydatid cyst, 28
Intracavitary drainage, 149
Intrapleural and extrapleural pneumothorax, 33
Intrathoracic sympathetic nerve tumors, 123

Jackman, R. J., and Buie, L. A. Anal fistula, 130
Jann, G. J., and Salle, A. J. Subtilin, 170
——, ——. ——., —— ——, ——. ——. Toxicity of subtilin to embryonic tissue, 170
Jarman, T. F., and Morris, G. J. Treatment of tuberculous empyema, 48
Jensen, C. R. Rhematic pneumonitis, 76
Jensen, K. A. BCG vaccination, 61
——, ——. ——., and Bindslev, J. Virulence of tubercle bacilli, 57
Jerram and Dell, D. Tuberculin testing, 7
Joly, H., and Carcopino, C. Results of thoracoplasty, 146
Jones, B. Surgical emphysema, 116
Josey, A. I., Trenis, J. W., and Kammer, W. F. Postpneumonic empyema, 119
Jouin, J.-P., and Buu-Hoi. Chemical inhibition of bacilli, 165
Joules, H., and Humphrey, J. H. Penicillin inhalation in pulmonary disease, 75
——, ——. See Fatti, L., *et al.*, 119
Juarez, R. G., and Oraindi, D. Prognosis in pleural effusions, 47
Juarrero, V. P., and Azparren, J. S. Toxic granulations in tuberculosis, 44

Kaiser, E. Tuberculous cervical lymph nodes, 51
Kaiser, S. J. See Heidelberger, M., *et al.*, 70
Kallos, P. Tuberculin allergy, 66
Kammer, W. F. See Josey, A. I., *et al.*, 119

Kane, L. W., and Brean, H. P. Pulmonary tuberculosis in medical students, 132
Kasius, R. V., and Pitney, Elizabeth H. Tuberculosis in major cities, 3
——, ——. ——., —— ——, ——. ——. Tuberculosis mortality in cities, 3
Katz, H. L., and Abel, H. A. Tuberculous meningitis, 45
——, ——. ——., —— Healy, M. J. Nontuberculous empyema, 118
Katz, H. W., and Russakoff, A. H. Bronchiectasis and dextrocardia, 113
Katz, J., Plunkett, R. E., and Thompson, Mary E. Tuberculosis in mental institutions, 6
Katz, S., and Reed, H. R. Radiology of pleural effusions, 47
Kay, E. B., and Meade, R. H., Jr. Penicillin in chronic pulmonary infections, 77
King, E. S. See Harrell, G. T., *et al.*, 67
Kinsey, F. R., and Herbut, P. A. Experimental Loeffler's syndrome, 80
Kinsman, J. M., Daniels, W. B., Cohen, S., McCracken, J. P., D'Alonso, C. A., Martin, S. P., and Kirby, W. M. M. Pneumonia, 72
Kirby, W. M. M. See Kinsman, J. M., *et al.*, 72
Klebsiella pneumoniae bacteremia, 129
Klosk, E., Bernstein, A., and Parsonnet, A. E. Cystic disease, 100
Knowlton, G. C., and Larrabee, M. G. Phrenic nerve response to lung inflation, 176
——, ——. ——., —— ——, ——. ——. Pulmonary volume receptors, 175
Kobacker, J. L., and Mehlin, G. B. Klebsiella pneumoniae bacteremia, 129
Koelsche, G. A. See Carryer, H. M., *et al.*, 105
Kornev, P. G. Surgery in bone tuberculosis, 154
Krafchik, L. F. Streptomycin in tuberculous meningitis, 167
Kramer, M., Comstock, G. W., and Stocklen, J. B. Resurvey of industrial plant, 5
Krause, G. R. Pulmonary infarcts, 96

Labesse, P. New method of spirometry, 173
Laird, R. Thoracoscopy, 32
Lambert, A. Partial claviculectomy, 148
Lander, F. P. L. Bronchiectasis and atelectasis, 181
Lange, K. Surgery for asthma, 106
——, ——. Treatment of pulmonary embolism, 94
Langer, L., and Silvestrini, H. Surgery for bronchiectasis, 114
Langevin, P. See D'Hour, H., *et al.*, 172
Larrabee, M. G., and Knowlton, G. C. Phrenic nerve response to lung inflation, 176
——, ——. ——., —— ——, ——. ——. Pulmonary volume receptors, 175
Laryngitis, tuberculous, Promin against, 51
Lasnier, E. P., and Cassinelli, J. F. Hydatid cyst, 84
Laymon, C. W., and Michelson, H. E. Classification of tuberculosis of skin, 151
Lecoeur, J., and Even, R. Ballooned, inert and residual cavities, 141
——, ——., —— ——, ——. Fever after refills, 142
Lehman, J. Metabolism of tubercle bacilli, 57
——, ——. Para-aminosalicyclic acid for tuberculosis, 39
——, ——. P-aminosolicyclic acid for tuberculosis, 169
Lehmann, G. O., and Prendiville, J. T. Flagellate in bronchiectasis, 181
Lemoine, J. M., Diacoumopoulos, A., and Paillas, J. Artificial pneumothorax, 32
"Leprosy bacilli," cultured, Disparity between Hansen's bacilli and, 157
Lerolle, M. See Biedermann, A., *et al.*, 132
Lesions, chronic, Chemistry of, 139
——, Nontuberculous, in mass X-ray surveys, 69
——, Pulmonary, in burns, 94
——, tuberculous, Tubercle bacilli in, 140
Leslie, Eleanor I. See Rosenthal, S. R., *et al.*, 163
Leslie, M. I. See Blahd, M., *et al.*, 12
Levenson, S. M., and Davidson, C. S. Pulmonary complications in burns, 94
——, ——. ——. See Finland, M., *et al.*, 94
Levin, Florence C. See Herman, M., *et al.*, 17
Lewis, H. P., and Feldman, D. J. Pleural effusions, 46
Lewis, I. Photofluorographic roll-film viewers, 6
——, ——., and Morgan, R. H. Stereoscopy, 133
Lewis-Jonsson, J. Tuberculosis from a cat, 45
Lieutier, H. See Cantonnet Blanch, P., *et al.*, 63

Lilienthal, J. L., Jr., Riley, R. L., Proemmel, D. D., and Franke, R. E. Alveolar and arterial oxygen pressure, 175
—, —. —., —, See Riley, R. L., *et al.*, 175
Linton, R. L. See Allen, A. W., *et al.*, 95
Liquid media, Growth of tubercle bacilli in, 54
Lipiodol reaction, 113
Liver puncture in sarcoidosis, 86
—, Tuberculomata of, 152
Lobar and segmental collapse of lung, 89
Lobe, right middle, Collapse of, 90
Local reactions to BCG, 163
Loeffler's syndrome, 77, 78
— —, Experimental, 80
Logan, J., and Edwards, P. W. Pneumoperitoneum, 35
Lower-lobe tuberculosis, 24, 25
Lung abscess, 77
—, Agenesis of, 68
— cancer, Biopsy of, 109
— inflation, Phrenic nerve response to, 176
—, Lobar and segmental collapse of, 89
—, Neuromuscular system of, 87
—, Segmental collapse of, 90
—, Syphilis of, 80, 81
— tumors, Primary, 107
—, wet, Traumatic, 98
Lymph node tuberculosis, 152
— nodes, cervical, Tuberculous, 51
— —, tracheobronchial, tuberculous, Perforating, 49
— —, Tuberculous, 50
Lymphoma, Malignant, 108
Lynch, J. P., and Holt, Rebecca A. Pulmonary actinomycosis, 83
—, —. —., — Strieder, J. W. Putrid empyema, 120

MacLeod, C. M. See Heidelberger, M., *et al.*, 70
Maclouf, A.-C., and Boulenger, P. Accelerated sensitization, 163
—, —. —. See Courcoux, A., *et al.*, 163
Magnin, M. F. Pneumoperitoneum with phrenic paralysis, 142
Mahon, G. S. Lipiodol reaction, 113
Major cities, Tuberculosis in, 3
Malignant lymphoma, 108
Mamma, Tuberculosis of, 53
Man, Avian tuberculosis in, 29
Manas, M. A. Acute silicosis, 102
Marital tuberculosis, 11
Marshak, A. Antibiotic from *Ramalina reticulata*, 165
Martin, D. S., and Hiatt, J. S., Jr. Moniliasis, 82
Martin, S. P. See Kinsman, J. M., *et al.*, 72
Mason, J. L. See Smith, C. R., *et al.*, 61
Mass X-ray surveys, Nontuberculous lesions in, 69
Material, Fatty, in bacteria and fungi, 157
May, H. B., and Floyer, M. A. Penicillin in bronchiectasis, 112
Maytum, C. K. See Carryer, H. M., *et al.*, 105
Mazaudier, M. T. See Perdigon, E., *et al.*, 164
Mazo, D. Ulcero-atelectatic syndrome, 23
McClosky, W. T. See Smith, M. I., *et al.*, 167
McCormick, W. E. Dust particle size, 103
McCracken, J. P. See Kinsman, J. M., *et al.*, 72
McDermott, W. See Bunn, P. A., *et al.*, 71
McDonald, J. B., and Ehrenpreis, B. Primary atypical pneumonia, 74
McDonald, J. R. See Moersch, H. J., *et al.*, 109
Meade, R. H., Jr., and Kay, E. B. Penicillin in chronic pulmonary infections, 77
Medeiros Q., S. Silicosis, 100
Media, liquid, Growth of tubercle bacilli in, 54
Mediastinal abscess, Anterior, 124
— emphysema, 122
— teratoma, 123
— tumors removed by surgery, 123
Mediastinum, Displacement of, 122
Medical students, Pulmonary tuberculosis in, 132
— —, Tuberculosis in, 8, 9
Medici, F. A., and Sampietro, R. Pneumonolysis in bilateral pneumothorax, 33
—, —. —. See Vaccarezza, R. F., *et al.*, 120
Medium, Culture, for tubercle bacillus, 55
Mehlin, G. B., and Kobacker, J. L. Klebsiella pneumoniae bacteremia, 129
Melick, D. W., and Spooner, Maryloo. Blood coagulation in pleural cavity, 117
Membranes, serous, Tuberculosis of, 49
Meningitis, Tuberculous, 45
—, —, Streptomycin in, 167
Menon, I. G. K. Tropical eosinophilia, 80
Mental deficiency, X-ray peculiarities in, 21
— institutions, Tuberculosis in, 6
Merrill, A. J., Warren, J. A., Stead, E. A., Jr., and Brannon, E. S. Circulation in penetrating chest wounds, 126
Mery, J. See Biedermann, A., *et al.*, 132

Metabolism of tubercle bacilli, 57, 156
Method of spirometry, New, 173
Meyer, J. Tubercle endotoxoid, 38
Michailov, F. A. Artificial pneumothorax, 31
Michelson, H. E., and Laymon, C. W. Classification of tuberculosis of skin, 151
Microörganisms, acid-fast, Atypical, 56
Miliary tuberculosis, 44
Miniature chest films, 6
Minimal tuberculosis, 19
Minneapolis, Principles of tuberculosis control in, 131
Minor, G. R., and White, M. L., Jr. Typhoid and salmonella infections, 87
Model, L. M. Pathophysiology of exudative tuberculosis, 22
Moersch, H. J., Tinney, W. S., and McDonald, J. R. Adenoma of bronchus, 109
Mollard, H., and Dumarest, H. Complications of pneumothorax, 143
Moniliasis, 82
Monod, R., and Cara, M. Pulmonary permeability, 176
Morgan, B. H., and Murphy, E. G. X-ray generator, 6
Morgan, R. H., and Lewis, I. Stereoscopy, 133
Moriyama, I. M., and Yerushalmy, J. Tuberculosis mortality, U. S. A. 1944, 3
Morris, G. J., and Jarman, T. F. Treatment of tuberculous empyema, 48
Mortality, Tuberculosis, in cities, 3
——, ——, —— nonwhites, 3
——, ——, U. S. A. 1944, 3
Morton, D. R., and Scott, O. B. Cyst of diaphragm, 121
Mosera, R., and Norman, J. Atelectasis after hemoptysis, 28
Mounier-Kuhn, P., and Dufourt, P. Chronic epituberculosis, 136
——, ——., Ollagnier, C., and Persillos, A. Hemoptysis and bronchoscopy, 182
——, ——. See Dufourt, A., *et al.*, 137
Munter, E. J. Pneumonic plague, 77
Murphy, E. G., and Morgan, B. H. X-ray generator, 6
M. tuberculosis, Granules of, 58
Mycolic acid, 58
Mycosis, Pulmonary, 81
Myers, J. A. Principles of tuberculosis control in Minneapolis, 131
——, ——. ——., Harrington, F. E., and Suarez, E. G. Tuberculosis in children, 12

Nasal septum, Tuberculosis of, 51
Nassau, E., and Roberts, J. C. Disseminated tuberculosis in children, 15
Nathan, D. A., and Dathe, R. A. Pericarditis following upper respiratory infection, 128
Navarro, L. G., and Roca, A. C. Pleural absorption of dyes, 68
Necrosis, Tuberculous, 162
Negative tuberculin reaction in active tuberculosis, 23
Negro, J. C. See Gomez, F. D., *et al.*, 31
Nelson, J. B. Endemic pneumonia in albino rat, 73
Nerve block in treatment of thoracic injuries, 126
——, Phrenic, paralysis, 30, 31
——, ——, response to lung inflation, 176
——, sympathetic, Intrathoracic, tumors, 123
Neuromuscular system of lung, 87
Newfoundland, Tuberculosis in, 131
Nichamin, S. J. Epidemic pleurodynia, 118
Nichols, A. C. See Zintel, H. A., *et al.*, 40
Nichols, D. R. See Heilman, F. R., *et al.*, 41
Nico, P., and Troisier, J. Tuberculosis in medical students, 9
Nielsen, G. Heparin and blood sedimentation, 44
Noguera, O. F., and Gotta, G. Pleural calcifications, 49
Nondisabling bronchiectasis, 110
Nontuberculous empyema, 118
—— lesions in mass X-ray surveys, 69
Nonwhites, Tuberculosis mortality in, 3
Norman, J., and Mosera, R. Atelectasis after hemoptysis, 28
Norris, C. M. See Durant, T. M., *et al.*, 73

Oleneva, T. N. Round foci, 136
Olivier, H.-R., and de Saint-Rat, L. Action of *Bacillus subtilis*, 170
Ollagnier, C. See Mounier-Kuhn, P., *et al.*, 182
Open pneumonolysis, 143
Operations, chest, Support for patients during, 146
Oraindi, D., and Juarez, R. G. Prognosis in pleural effusions, 47
Organisms, Acid-fast, in gastric contents, 43
Ortuzar, R., Croxatto, R., and Cruzat, M. Pulmonary mycosis, 81
Osipov, I. N., and Soboleva, A. S. Tuberculous lymph nodes, 50
Osteoarthritis, Tuberculous, 52
Oxidized gauze in thoracoplasties, 148
Oxygen pressure, Alveolar and arterial, 175

Pacific, Southwest, Asthma in, 105
Packard, E. N. Miliary tuberculosis, 44
Pagel, W., and Hall, S. Congenital tuberculosis, 149
Pages, J. See Delaunay, A., *et al.*, 139
Paillas, J., and Bariéty, M. Perforation of tension cavity, 136
——, ——., —— ——, ——. Tubercle bacilli in bronchoscopic aspirations, 159
——, ——., —— ——, ——. X-ray appearance of draining bronchi, 27
——, ——. See Lemoine, J. M., *et al.*, 32
Paillas, P. See Biedermann, A., *et al.*, 132
Pain in chest, 124
Palmer, C. E. Histoplasmin reactors, 7
——, ——. ——., and Aronson, J. D. BCG vaccination in Indians, 63
——, ——. ——., —— Zwerling, H. B. Pulmonary calcifications, 177
P-aminosalicylic acid against various bacteria, 169
—— ——, Clinical experiences with, 169
—— —— for tuberculosis, 169
Para-aminosalicylic acid for tuberculosis, 39
Paralysis, Phrenic, 141
——, —— nerve, 30, 31
——, ——, Pneumoperitoneum with, 142
Parfenova, J. P. Bone tuberculosis, 152
Parique and Tulou. Marital tuberculosis, 11
Parsonnet, A. E. See Klosk., *et al.*, 100
Partial claviculectomy, 148
Particle size, Dust, 103
Patalano, A., and Soubrie, A. J. Congo red test, 171
Patch tests, Tuberculin, 7
Pathophysiology of exudative tuberculosis, 22
Patients during chest operations, Support for, 146
——, tuberculosis, Rehabilitation of, 30
Patt, H. M., Tobias, J. M., Swift, M. N., Postel, S., and Gerard, R. W. Hemodynamics in phosgene poisoning, 176
Peck, W. M. Bed-rest, 29
Peculiarities, X-ray, in mental deficiency, 21
Penetrating chest wounds, Circulation in, 126
Penicillin, Extrapleural pneumothorax and, 145
——, History of, 164
—— in acute empyema, 119
—— —— bronchial disease, 112
—— —— bronchiectasis, 112
—— —— chronic pulmonary infections, 77
—— —— pulmonary resections, 39
—— inhalation in pneumonia, 72
—— —— —— pulmonary disease, 75
——, Tuberculous abscess following, 155
Perdigon, E., Bouquet, F., Mazaudier, M. T., and Godard, F. Antagonistic growth substances, 164
Perforating tuberculous tracheobronchial lymph nodes, 49
Perforation of tension cavity, 136
Pericarditis following upper respiratory infection, 128
Perkins, M. E. See Halbert, P., *et al.*, 69
Permeability, Pulmonary, 176
Pernicious anemia and tuberculosis, 139
Peroncini, J. See Bence, A., *et al.*, 109
Persillos, A. See Mounier-Kuhn, P., *et al.*, 182
Peters, J. T. History of penicillin, 164
Petrik, F. G. Atypical acid-fast microörganisms, 56
Pfuetze, K. H. See Hinshaw, H. C., *et al.*, 166
Pharmacology of streptomycin, 40, 41
Phillips, Eileen, and Stewart, C. A. Pulmonary rarefaction, 98
Phosgene poisoning, Hemodynamics in, 176
Photofluorographic roll-film viewers, 6
—— survey, 133
Photoroentgenography in tuberculosis prophylaxis, 4
Phrenic nerve paralysis, 30, 31
—— —— response to lung inflation, 176
—— paralysis, 141
—— ——, Pneumoperitoneum with, 142
Physical diagnosis, 69
Piaggio, A. A., and Purriel, P. Incipient pulmonary tuberculosis, 19
Piaggio Blanco, R. A., and Canabal, E. J. Insufflated hydatid cyst, 28
Pilcher, R. Bronchiectasis, 114
Pilling, M. A. See Hirshfeld, J. W., *et al.*, 118
Pitney, Elizabeth H., and Kasius, R. V. Tuberculosis in major cities, 3
——, ——. ——., —— ——, ——. ——. Tuberculosis mortality in cities, 3
Pittaluga, R. E. M. Syphilis of lung, 81
Pitts, R. F. Respiratory centre, 173
Plague, Pneumonic, 77
Pleura, Echinococcus of, 121
——, Hydatid disease of, 120
Pleural absorption of dyes, 68
—— calcifications, 49
—— cavity, Blood coagulation in, 117
—— effusions, 46
—— ——, Bilateral, 47
—— ——, Prognosis in, 47
—— ——, Radiology of, 47
Pleurisy, Dry, 118
Pleurodynia, Epidemic, 118
Plombage, 36

Plum, P., and Poulsen, J. E. Blood prothrombin in tuberculosis, 25
Plunkett, R. E. See Katz, J., *et al.*, 6
Pneumococcal hemolysin, 69
Pneumococci and polysaccharides, Antibodies to, 70
——, Antibodies against, 71
Pneumonia, 71, 72
——, atypical, Primary, 74
——, Electrocardiogram in, 73
——, Endemic, in albino rat, 73
—— in aged, 72
——, Influenza, 73
——, Penicillin inhalation in, 72
——, Streptococcus, 76
Pneumonic plague, 77
Pneumonitis, Rheumatic, 76
Pneumonolysis, Extrapleural, 145
—— in bilateral pneumothorax, 33
——, Open, 143
Pneumoperitoneum, 34, 35
—— against hemoptysis, 36
——, Electrocardiogram in, 36
—— with phrenic paralysis, 142
Pneumothorax, Artificial, 31, 32
——, bilateral, Pneumonolysis in, 33
——, Complications of, 143
——, Cure of early bronchiectasis by, 180
——, Duration of, 142
——, Empyema in, 142
——, Extrapleural, 144, 145
——, ——, and penicillin, 145
—— following bronchoscopy, 116
——, Intrapleural and extrapleural, 33
Poisoning, Beryllium, 104
——, phosgene, Hemodynamics in, 176
Pollitzer, G. See Vaccarezza, R. F., *et al.*, 120
Polysaccharides, Antibodies to pneumococci and, 70
Poppe, J. K. Treatment of pulmonary actinomycosis, 182
Postel, S. See Patt, H. M., *et al.*, 176
Postoperative pulmonary collapse, 93
Postpneumonic empyema, 119
Postpneumothorax bronchospirometry, 34
Poulsen, J. E., and Plum, P. Blood prothrombin in tuberculosis, 25
Pregnancy, Tuberculosis and, 22
Premenstrual asthma, 107
—— fever in tuberculosis, 137
Prendiville, J. T., and Lehmann, G. O. Flagellate in bronchiectasis, 181
Preservation of tubercle bacilli, 159
Pressure, gas, Alveolar, 175
——, oxygen, Alveolar and arterial, 175
Price, A. H., and Teplick, G. Bullous emphysema, 100
Prickman, L. E. See Carryer, H. M., *et al.*, 105
Primary atypical pneumonia, 74
—— infection, 135
—— lung tumors, 107
—— thoracoplasty, 146
—— tuberculosis, 15, 17
—— —— in adults, 16, 134, 135
—— —— of conjunctiva, 151
—— ——, Treatment of, 15
Prisoners of war, Tuberculosis among, 10, 11
Proemmel, D. D. See Lilienthal, J. L., Jr., *et al.*, 175
——, ——. ——. See Riley, R. L., *et al.*, 175
Prognosis in pleural effusions, 47
Program, Tuberculosis Association, 131
Promin against tuberculous laryngitis, 51
—— in rat tuberculosis, Streptomycin and, 167
Prophylaxis, tuberculosis, Photoroentgenography in, 4
Prostitutes, Tuberculosis in, 133
Prothrombin, Blood, in tuberculosis, 25
Pulmonary actinomycosis, 83
—— ——, Treatment of, 182
—— adenomatosis, 108
—— and endobronchial tuberculosis, 137
—— calcifications, 177, 178
—— —— and age, 178
—— collapse, Experimental, 92
—— ——, Postoperative, 93
—— complications in burns, 94
—— disease, Penicillin inhalation in, 75
—— embolism, 95
—— ——, Treatment of, 94
—— Hodgkin's disease, 110
—— infarcts, 96
—— infections, chronic, Penicillin in, 77
—— lesions in burns, 94
—— mycosis, 81
—— permeability, 176
—— rarefaction, 98
—— resection in tuberculosis, 38
—— resections, Penicillin in, 39
—— segments, 88
—— thrombosis, 97
—— tuberculosis in medical students, 132
—— ——, Incipient, 19
—— ——, Symptoms in, 138
—— ventilation and anoxemia, 174
—— volume receptors, 175

Puncture, Liver, in sarcoidosis, 86
Purriel, P., and Piaggio, A. A. Incipient pulmonary tuberculosis, 19
Putrid empyema, 120

Quist, G. Chest wounds, 126

Rabich-Shchebro, V. A., and Ignatowskaja, N. N. Phrenic nerve paralysis, 30
Radiology of pleural effusions, 47
Raffel, S. Immunology of tuberculosis, 64
Railroad employees, Fluoroscopy in, 132
Ramalina reticulata, Antibiotic from, 165
Rarefaction, Pulmonary, 98
Rat, albino, Endemic pneumonia in, 73
—— tuberculosis, Streptomycin and promin in, 167
Reaction, Lipiodol, 113
——, tuberculin, Negative, in active tuberculosis, 23
Reactions, Anaphylactic, to Congo red, 130
——, Local, to BCG, 163
Reactivity, tuberculin, Cellular transfer of, 162
Reactors, Histoplasmin, 7
Rebora, F. Phrenic paralysis, 141
Rebull Richieri, V., and Gines, A. R. BCG, 62
Receptors, Pulmonary volume, 175
Recovery from hemolytic staphylococcus aureus bacteremia, 129
Reed, H. R., and Katz, S. Radiology of pleural effusions, 47
Refills, Fever after, 142
Rehabilitation of tuberculosis patients, 30
Reid, H. Extrapleural pneumothorax, 144
Relaxing thoracoplasty, 37
Renal tuberculosis, 52
Resection, Pulmonary, in tuberculosis, 38
Resections, pulmonary, Penicillin in, 39
Residual cavities, Ballooned, inert and, 141
Respiration in cardiac dyspnea, 176
—— of tubercle bacilli, 156
Respiratory air-flow, 173
—— centre, 173
—— infection, upper, Pericarditis following, 128
—— infections, Upper, 179, 180
Results of thoracoplasty, 146
Resurvey of industrial plant, 5
Revision thoracoplasty, 147
Rey, J. See Vaccarezza, R. F., *et al.*, 145
Rey, J. C. See Bence, A., *et al.*, 109
Rheumatic pneumonitis, 76
Rheumatism, Tuberculous, 46
Rhoads, J. E. See Zintel, H. A., *et al.*, 40
Richards, W. F. Tuberculosis in children, 13
Right middle lobe, Collapse of, 90
—— upper lobe bronchus, Anatomy of, 172
Riley, R. L., Lilienthal, J. L., Jr., Proemmel, D. D., and Franke, R. E. Alveolar gas pressure, 175
——, ——. ——. See Lilienthal, J. L., Jr., *et al.*, 175
Rist, M. E. Tuberculosis in medical students, 8
Ritter, W. L., and Bovard, P. G. Hazards in fire-brick industry, 104
Ritvo, M. See Finland, M., *et al.*, 94
Robbins, L. L., and Hale, C. H. Collapse of right middle lobe, 90
——, ——. ——., —— ——, ——. ——. Lobar and segmental collapse of lung, 89
——, ——. ——., —— ——, ——. ——. Segmental collapse of lung, 90
Roberts, J. C., and Nassau, E. Disseminated tuberculosis in children, 15
Robin, I. G. Promin against tuberculous laryngitis, 51
Robinson, B. See Heidelberger, M., *et al.*, 70
Robinson, H. J., and Stebbins, R. B. Streptomycin in body fluids, 168
——, ——. ——. See Stebbins, R. B., *et al.*, 168
Roca, A. C., and Navarro, L. G. Pleural absorption of dyes, 68
Rodriguez Folgueras, J. M., and Toulet, I. P. Premenstrual asthma, 107
Rolland, J. Complications of thoracoplasty, 146
Roll-film viewers, Photofluorographic, 6
Rosenthal, S. R., Blahd, Margery, and Leslie, Eleanor I. BCG vaccination, 163
——, ——. ——. See Blahd, M., *et al.*, 12
Round foci, 136
Russakoff, A. H., and Katz, H. W. Bronchiectasis and dextrocardia, 113
Rutstein, D. D. See Thomson, K. J., *et al.*, 73

Sakula, J. See Fatti, L., *et al.*, 119
Salle, A. J., and Jann, G. J. Subtilin, 170
——, ——. ——., —— ——, ——. ——. Toxicity of subtilin to embryonic tissue, 170
Salmonella infections, Typhoid and, 87
Sampietro, R. Pulmonary resection in tuberculosis, 38
——, ——., and Medici, F. A. Pneumonolysis in bilateral pneumothorax, 33
Samson, P. C. Thoracic wounds, 125
——, ——. ——., Major. See Buford, T. H., Major, *et al.*, 129

Sanatoria, Finnish, 6
Sanjuan Nadal, H. Granules of *M. tuberculosis*, 58
Sarcoidosis, Boeck's, 86
——, Liver puncture in, 86
Scadding, J. G. Dry pleurisy, 118
Scandoglio, J. J. Perforating tuberculous tracheobronchial lymph nodes, 49
Scandroglio, I. I. Tuberculosis of mamma, 53
Scatchard, G. N., and Duszynski, Diana Olga. Miniature chest films, 6
Schaub, K. See Geiser, P., *et al.*, 72
Schenkein, E. L. See Walter, A. M., *et al.*, 71
Schjelderup, H., and Birkhaug, K. Hematology in experimental tuberculosis, 66
Schleicher, E. M. Pernicious anemia and tuberculosis, 139
Schlittler, E. Tuberculosis of tonsils, 152
Schlumberger, H. G. Mediastinal teratoma, 123
Schmidt, E. A. Pneumoperitoneum, 34
Schuman, C. Breath sounds, 43
Sciuto, J. A. Emphysema, 99
Scott, H. W., Jr., and Gross, R. E. Esophageal atresia and tracheo-esophageal fistula, 124
Scott, O. B., and Morton, D. R. Cyst of diaphragm, 121
Screens, Fluoroscopic, 134
Sedimentation, blood, Heparin and, 44
Segmental collapse of lung, 90
—— —— —— ——, Lobar and, 89
Segments, Pulmonary, 88
Selikoff, I. J. Congo red test, 171
——, ——. ——., and Bernstein, I. J. Anaphylactic reactions to Congo red, 130
Seltzer, I., and Goldfarb, A. A. Primary tuberculosis of conjunctiva, 151
Sensitivity, tuberculin, Persistence of, 162
Sensitization, Accelerated, 163
Septum, nasal, Tuberculosis of, 51
Serous membranes, Tuberculosis of, 49
Sheldon, W. Tuberculous rheumatism, 46
Sierra, R. P. Thoracopneumonolysis, 37
Sievers, O. P-aminosalicylic acid against various bacteria, 169
Silicosis, 100, 102
——, Acute, 102
——, Workmen's compensation for, 103
Silverman, L. Respiratory air-flow, 173
Silvestrini, H., and Langer, L. Surgery for bronchiectasis, 114
Simmonds, F. A. H. Pneumoperitoneum, 34
Singletary, W. V., and Eichwald, M. Loeffler's syndrome, 78
Skin, Tuberculosis of, 150
——, —— ——, Classification of, 151
Smart, J. Agenesis of lung, 68
Smith, C. R., Urabec, J. H., and Mason, J. L. Air-borne tubercle bacilli, 61
Smith, M. I., McClosky, W. T., and Emmart, W. T. Streptomycin and promin in rat tuberculosis, 167
Smyth, C. J., and Billingslea, T. H. Lung abscess, 77
Soboleva, A. S., and Osipov, I. N. Tuberculous lymph nodes, 50
Sokalchuk, A. J. See Durant, T. M., *et al.*, 73
Sokol, D. D. See Fox, T. T., *et al.*, 29
Soldiers, discharged, Chest examinations in, 132
Solem, J. Negative tuberculin reaction in active tuberculosis, 23
Soltya, N. A., and Alexander, A. E. Surface-active substances and tubercle bacilli, 159
Soubrie, A. J., and Patalano, A. Congo red test, 171
Soukup, E. Foreign bodies, 115
Soulas, A. Bronchoscopic treatment, 139
Southwell, N. Penicillin in bronchial disease, 112
Southwest Pacific, Asthma in, 105
Spain, D. M., and Handler, B. J. Cor pulmonale, 127
Spector, H. I. Loeffler's syndrome, 77
Spickers, W. Support for patients during chest operations, 146
Spirometry, New method of, 173
Spleen, Calcifications in, 178
Spooner, Maryloo, and Melick, D. W. Blood coagulation in pleural cavity, 117
Sporer, A. Renal tuberculosis, 52
Spread of dye in allergy, 65
Sprick, Marian G., and Towey, J. W. Tubercle bacilli in gastric contents, 43
Sputum, Demonstration of tubercle bacilli in, 43
Stalker, W. W. Hazards in fire-brick industry, 103
Ställberg-Stenhagen, S., and Stenhagen, E. Mycolic acid, 58
Staphylococcus aureus bacteremia, hemolytic, Recovery from, 129
Starr, M. P. See Hampton, S. F., *et al.*, 107
Staub, H. See Geiser, P., *et al.*, 72
Stead, E. A., Jr. See Merrill, A. J., *et al.*, 126

Stebbins, R. B., and Robinson, H. J. Streptomycin in body fluids, 168
—, —. —., Graessle, O. E., and Robinson, H. J. Absorption and excretion of streptomycin, 168
Steen, P. Lower-lobe tuberculosis, 25
Steinhausen, T. B., and Fine, A. Nondisabling bronchiectasis, 110
Stenhagen, F., and Ställberg-Stenhagen, S. Mycolic acid, 58
Stereoscopy, 133
Stewart, C. A. Tuberculosis survey, 5
—, —. —., and Phillips, Eileen. Pulmonary rarefaction, 98
Stimulation, Growth, of tubercle bacilli, 160
Stocklen, J. B. See Kramer, M., *et al.*, 5
Stone, C. T. Boeck's sarcoidosis, 86
Stoner, R. E., and Corper, H. J. Culture of tubercle bacilli, 160
—, —. —., — —, —. —. Diagnostic culture of tubercle bacilli, 160
Strains of tubercle bacilli, Streptomycin resistant, 168
Streptococcus pneumonia, 76
Streptomycin, Absorption and excretion of, 168
— and promin in rat tuberculosis, 167
— — streptothricin, Cytotoxicity of, 168
— in body fluids, 168
— — tuberculosis, 166
— — tuberculous meningitis, 167
— — urinary tract tuberculosis, 53
—, Inactivation of, 168
—, Pharmacology of, 40, 41
— resistant strains of tubercle bacilli, 168
— toxicity, 42
Streptothricin, Cytotoxicity of streptomycin and, 168
Strieder, J. W., and Lynch, J. P. Putrid empyema, 120
Students, medical, Pulmonary tuberculosis in, 132
—, —, Tuberculosis in, 8, 9
Suarez, E. G. See Myers, J. A., *et al.*, 12
Suarez, H. See Ugon, V. A., *et al.*, 121
Submerged growth of tubercle bacilli, 159
Subtilin, 170
—, Toxicity of, to embryonic tissue, 170
Support for patients during chest operations, 146
Surface-active substances and tubercle bacilli, 159
Surgery, chest, Instrument tray for, 146
— for asthma, 106
— — bronchiectasis, 114
Surgery in bone tuberculosis, 153
—, Mediastinal tumors removed by, 123
Surgical emphysema, 116
Survey, Photofluorographic, 133
—, Tuberculosis, 4, 5
Surveys, Abnormal X-ray findings in, 138
—, X-ray, mass, Nontuberculous lesions in, 69
Suter, F. Renal tuberculosis, 52
Sutliff, W. D. See Walter, A. M., *et al.*, 71
Swift, M. N. See Patt, H. M., *et al.*, 176
Swine, Tuberculosis in, 170
Sympathetic nerve tumors, Intrathoracic, 123
Symptoms in pulmonary tuberculosis, 138
— — tuberculosis, 20
Syndrome, Loeffler's, 77, 78
—, —, Experimental, 80
—, Ulcero-atelectatic, 23
Syphilis of lung, 80, 81

Tension cavity, Perforation of, 136
Teplick, G., and Price, A. H. Bullous emphysema, 100
Teratoma, Mediastinal, 123
Test, Congo red, 171
Testing, Tuberculin, 7
Tests, Coccidioidin, 82
—, patch, Tuberculin, 7
Therapy, collapse, Indications for, 140
Thomas, D., and Barrett, M. R. Hydatid cyst, 85
Thomas, G. O., and Hawkins, S. F. Lower-lobe tuberculosis, 24
Thompson, G. S. See Hampton, S. F., *et al.*, 107
Thompson, Mary E. See Katz, J., *et al.*, 6
Thompson, S. E. See Herman, M., *et al.*, 17
Thomson, K. J., Rutstein, D. D., Tolmach, D. M., and Walker, W. H. Electrocardiogram in pneumonia, 73
Thoracic injuries, Nerve block in treatment of, 126
— wounds, 125
Thoracoplasties, Oxidized gauze in, 148
Thoracoplasty, Complications of, 146
— New incision for, 147
—, Primary, 146
—, Relaxing, 37
—, Results of, 146
—, Revision, 147
Thoracopneumonolysis, 37
Thoracoscopy, 32
Thrombosis, Pulmonary, 97
Tinney, W. S. See Moersch, H. J., *et al.*, 109

Tissue, embryonic, Toxicity of subtilin to, 170
Tobias, J. M. See Patt, H. M., *et al.*, 176
Tolmach, D. M. See Thomson, K. J., *et al.*, 73
Tonsils, Tuberculosis of, 152
Toulet, I. P., and Rodriguez Folgueras, J. M. Premenstrual asthma, 107
Towey, J. W., and Sprick, Marian G. Tubercle bacilli in gastric contents, 43
Toxic granulations in tuberculosis, 44
Toxicity of subtilin to embryonic tissue, 170
——, Streptomycin, 42
Tracheobronchial lymph nodes, tuberculous, Perforating, 49
Tracheo-esophageal fistula, Esophageal atresia and, 124
Transfer, Cellular, of tuberculin reactivity, 162
Traumatic wet lung, 98
Travis, O. T., Harrell, G. T., and King, E. S. Tuberculosis and trichinella, 67
Tray, Instrument, for chest surgery, 146
Treatment, Bronchoscopic, 139
——, Diasone, 40
—— of bronchiectasis, 111
—— —— primary tuberculosis, 15
—— —— pulmonary actinomycosis, 182
—— —— —— embolism, 94
—— —— thoracic injuries, Nerve block in, 126
—— —— tuberculous empyema, 48
Trenchard, H. J. Symptoms in tuberculosis, 20
Trenis, J. W. See Josey, A. I., *et al.*, 119
Trent, J. C. Aneurysm of innominate artery, 128
Trichinella, Tuberculosis and, 67
Troisier, J., and Nico, P. Tuberculosis in medical students, 9
Tropical eosinophilia, 79, 80
Tubercle bacilli, Air-borne, 61
—— ——, Alkylresorcinols on, 59
—— —— and urea, 59
—— ——, Culture of, 160
—— ——, Demonstration of, in sputum, 43
—— ——, Diagnostic culture of, 160
—— ——, Dissociation of, 158
—— ——, Growth of, in liquid media, 54
—— ——, —— rate of, 56
—— ——, —— stimulation of, 160
—— —— in bronchoscopic aspirations, 159
—— —— —— gastric contents, 43
—— —— —— tuberculous lesions, 140
—— ——, Metabolism of, 57, 156
—— ——, Preservation of, 159
—— ——, Respiration of, 156
—— ——, Streptomycin resistant strains of, 168
Tubercle bacilli, Submerged growth of, 159
—— ——, Surface-active substances and, 159
—— ——, Virulence of, 57
—— ——, Vole, 161
—— bacillus, Culture medium for, 55
—— endotoxoid, 38
Tuberculin allergy, 66
—— patch tests, 7
—— reaction, Negative, in active tuberculosis, 23
—— reactivity, Cellular transfer of, 162
—— sensitivity, Persistence of, 162
—— testing, 7
Tuberculomata of liver, 152
Tuberculosis, active, Negative tuberculin reaction in, 23
—— among prisoners of war, 10, 11
—— and pregnancy, 22
—— —— trichinella, 67
—— Association program, 131
——, Avian, in man, 29
——, Beds for, 3
——, Blood prothrombin in, 25
——, Bone, 152
——, ——, Surgery in, 153
——, Cardiospasm in, 139
——, Cattle, in U.S.A., 67
——, Congenital, 149
—— control division, 2
—— —— in Minneapolis, Principles of, 131
——, Disseminated, in children, 15
——, experimental, Hematology in, 66
——, exudative, Pathophysiology of, 22
—— from a cat, 45
——, Genetic factor in, 17
——, Genito-urinary, 154, 155
——, Global distribution of, 2
——, Hepatitis and, 28
——, Immunology of, 64, 65
—— in children, 12, 13, 14
—— —— Denmark, 1
—— —— hamsters, 161
—— —— major cities, 3
—— —— medical students, 8, 9
—— —— mental institutions, 6
—— —— Newfoundland, 131
—— —— prostitutes, 133
—— —— swine, 170
——, Inoculation, 150
——, Lower-lobe, 24, 25
——, Lymph node, 152
——, Marital, 11
——, Miliary, 44
——, Minimal, 19

Tuberculosis mortality in cities, 3
—— —— —— nonwhites, 3
—— ——, U. S. A. 1944, 3
—— of mamma, 53
—— —— nasal septum, 51
—— —— serous membranes, 49
—— —— skin, 150
—— —— ——, Classification of, 151
—— —— tonsils, 152
—— —— uterus, 154
——, P-aminosalicylic acid for, 169
——, Para-aminosalicylic acid for, 39
—— patients, Rehabilitation of, 30
——, Pernicious anemia and, 139
——, Premenstrual fever in, 137
——, Primary, 15, 17
——, ——, in adults, 16, 134, 135
——, ——, of conjunctiva, 151
——, ——, Treatment of, 15
—— prophylaxis, Photoroentgenography in, 4
——, Pulmonary and endobronchial, 137
——, ——, in medical students, 132
——, ——, Incipient, 19
——, —— resection in, 38
——, ——, Symptoms in, 138
——, rat, Streptomycin and promin in, 167
——, Renal, 52
——, Streptomycin in, 166
—— survey, 4, 5
——, Symptoms in, 20
——, Toxic granulations in, 44
——, urinary tract, Streptomycin in, 53
——, Ventricular deflections in, 29
Tuberculostatic substances, 165
Tuberculous abscess following penicillin, 155
—— arthritis, 153
—— bronchitis, 25
—— cervical lymph nodes, 51
—— empyema, Treatment of, 48
—— laryngitis, Promin against, 51
—— lesions, Tubercle bacilli in, 140
—— lymph nodes, 50
—— meningitis, 45
—— ——, Streptomycin in, 167
—— necrosis, 162
—— osteoarthritis, 52
—— rheumatism, 46
—— tracheobronchial lymph nodes, Perforating, 49
Tulou and Parique. Marital tuberculosis, 11
Tumor, cell, Alveolar, 107
Tumors, lung, Primary, 107
——, Mediastinal, removed by surgery, 123
——, sympathetic nerve, Intrathoracic, 123
Typhoid and salmonella infections, 87

Ugon, V. A., Victorica, A., and Suarez, H. Echinococcus of pleura, 121
Ulcero-atelectatic syndrome, 23
U. S. A. 1944, Tuberculosis mortality, 3
——, Cattle tuberculosis in, 67
Upper respiratory infection, Pericarditis following, 128
—— —— infections, 179, 180
Urabec, C. R. See Smith, C. R., *et al.*, 61
Urea, Tubercle bacilli and, 59
Urinary tract tuberculosis, Streptomycin in, 53
Ustvedt, H. J. Primary tuberculosis in adults, 134
Uterus, Tuberculosis of, 154

Vaccarezza, O. A. Lymph node tuberculosis, 152
——, ——. ——. See Vaccarezza, R. F., *et al.*, 145
Vaccarezza, R. F., and Dutrey, J. Genetic factor in tuberculosis, 17
——, ——. ——., Pollitzer, G., and Medici, M. A. Hydatid disease of pleura, 120
——, ——. ——., Vaccarezza, O. A., and Rey, J. Extrapleural pneumothorax, 145
Vaccination, BCG, 61, 163
——, ——, in Indians, 63
Valdivieso D., R. Treatment of bronchiectasis, 111
Valle, A. B., and White, M. L. Penicillin in pulmonary resections, 39
Vallentin, G. Clinical experiences with p-aminosalicylic acid, 169
Van Allen, W. W. Fluoroscopic screens, 134
van Buchem, F. S. P. Liver puncture in sarcoidosis, 86
van Deinse, F. Bovine type bacilli, 158
——, ——. Changing virulence, 155
——, ——. Dissociation of tubercle bacilli, 158
van den Eeckhout, H. Primary infection, 135
Van Ordstrand, H. S., Hughes, R., DeNardi, J. M., and Carmodi, M. G. Beryllium poisoning, 104
Velasco, J. M. Rehabilitation of tuberculosis patients, 30
Vendrely, R. See Delaunay, A., *et al.*, 139
Ventilation, Pulmonary, and anoxemia, 174
Ventricular deflections in tuberculosis, 29
Victorica, A. See Ugon, V. A., *et al.*, 121
Vineberg, A. M., and Aronovitch, M. Anterior mediastinal abscess, 124
Virulence, Changing, 155
—— of tubercle bacilli, 57
Vitez, M. Malignant lymphoma, 108

Vogt, J. H. Erythema nodosum, 128
Vole tubercle bacilli, 161
Volume, Pulmonary, receptors, 175

Waksman, S. A. See Geiger, W. B., *et al.*, 168
Walker, W. H. See Thomson, K. J., *et al.*, 73
Wallace, H. J. Tuberculous lymph nodes, 50
Wallach, K., and Zeman, F. D. Pneumonia in aged, 72
Wallgren, A. Treatment of primary tuberculosis, 15
Walter, A. M., Schenkein, E. L., and Sutliff, W. D. Antibodies against pneumococci, 71
War, prisoners of, Tuberculosis among, 10, 11
Warren, J. A. See Merrill, A. J., *et al.*, 126
Weber, F. J. Global distribution of tuberculosis, 2
Weil, J., and Bernard, E. Duration of pneumothorax, 142
Welch, H. J. See Brantigan, O. C., *et al.*, 37
Welding hazards, 104
Wet lung, Traumatic, 98
White, M. L., Jr., and Minor, G. R. Typhoid and salmonella infections, 87
White, M. L., and Valle, A. B. Penicillin in pulmonary resections, 39
Whorton, C. M., and Chapman, C. B. Miliary tuberculosis, 44
Wiles, F. J. See Dormer, B. A., *et al.*, 80
Wiley, M. M. See Zintel, H. A., *et al.*, 40
Williston, Elizabeth H., and Youmans, G. P. Streptomycin resistant strains of tubercle bacilli, 168
Wilson, D. A., and Baker, H. Experimental pulmonary collapse, 92
Wine, M. B. See Hampton, S. F., *et al.*, 107
Winkenwerder, W. L. Asthma in Southwest Pacific, 105
Wood, F. C., and Hodes, P. J. Tropical eosinophilia, 79
Woolley, J. S. See Bobrowitz, I. D., *et al.*, 112
Workmen's compensation for silicosis, 103
Wounds, Chest, 126
——, ——, penetrating, Circulation in, 126
——, Thoracic, 125
Wright, D. O., and Gold, E. M. Loeffler's syndrome, 78

Xalabarder, D. Neuromuscular system of lung, 87
X-ray appearance of draining bronchi, 27
—— diagnosis in bronchiectasis, 113
—— findings, Abnormal, in surveys, 133
—— generator, 6
—— peculiarities in mental deficiency, 21
—— surveys, mass, Nontuberculous lesions in, 69

Yates, J. Acid-fast organisms in gastric contents, 43
Yegian, D., Budd, V., and Middlebrook, G. Biological changes in *M. ranae*, 60
Yerushalmy, J. Tuberculosis mortality in nonwhites, 3
——, ——., and Moriyama, I. M. Tuberculosis mortality, U. S. A. 1944, 3
Youmans, G. P. Growth rate of tubercle bacilli, 56
——, ——. ——., and Williston, Elizabeth H. Streptomycin resistant strains of tubercle bacilli, 168
Young, D. Upper respiratory infections, 180

Zeman, F. D., and Wallach, K. Pneumonia in aged, 72
Zintel, H. A., Flippin, H. E., Nichols, A. C., Wiley, M. M., and Rhoads, J. E. Pharmacology of streptomycin, 40
Zwerling, H. B., and High, R. H. Pulmonary calcifications and age, 178
——, ——. ——., —— Palmer, C. E. Pulmonary calcifications, 177
——, ——. ——. See High, R. H., *et al.*, 178

ALUM ROCK SANATORIUM

SAN JOSE, CALIFORNIA

Telephone: MAYFAIR 321

A non-profit Sanatorium for the treatment of Tuberculosis and other diseases of the chest. Located in the eastern foothills overlooking Santa Clara Valley.

Medical Director
Buford H. Wardrip, M.D.
Associate Medical Director
C. G. Scarborough, M.D.

Consultants:

Harold Guyon Trimble, M.D., Oakland
Cabot Brown, M.D., San Francisco
J. Lloyd Eaton, M.D., Oakland
Glenroy N. Pierce, M.D., San Francisco
Gerald L. Crenshaw, M.D., Oakland
Ina Gourley, M.D., Oakland

The Original is Worth $35.00

Doctors Are Christmas Seal Collectors

A hobby in which many members of the medical profession find relaxation. You may indulge at home or office — any place you have the small kit of necessary material. Inexpensive too — a collection of Christmas Seals finds a ready market.

Start now by writing the Collectors Service,
National Tuberculosis Association, 1790 Broadway, New York 19, N. Y.

NOTICE TO SUBSCRIBERS AND CONTRIBUTORS

THE AMERICAN REVIEW OF TUBERCULOSIS is published by the National Tuberculosis Association and issued monthly about the 1st of the month. THE AMERICAN REVIEW OF TUBERCULOSIS is the Official Journal of the American Trudeau Society. A volume includes six numbers and begins with the January and July numbers.

Subscriptions: The subscription price of the REVIEW is $8.00 for the calendar year. Checks should be made payable to Collier Platt, Treasurer.

Character of the Review: The REVIEW consists of two main parts, namely, (1) original articles and (2) abstracts. The original part is published monthly, its size depending upon the amount of manuscript in hand. Abstracts will appear as the amount of material warrants. Each part will be paged separately, to permit permanent separate binding upon the completion of a volume.

Manuscripts. The REVIEW invites the submission of manuscripts on any phase of tuberculosis and related subjects of interest to medical practitioners and students and workers in tuberculosis and public health. Articles are accepted for publication with the understanding that they are contributed solely to THE AMERICAN REVIEW OF TUBERCULOSIS.

Manuscripts should be sent to the office of the Editor, Dr. Max Pinner, 364 Fourteenth St., Oakland 12, California. They should be in English, typewritten on one side of the page only and with wide spacing and margins. They should be mailed flat and transmitted by first-class mail with postage for return if not available. Authors should exercise particular care in the preparation, notation and description of figures, charts and tables.

The publishers will not be responsible for manuscripts, illustrations, etc., lost in transit. In order to save expense for authors' corrections in proof, manuscript should be carefully revised by the author before submission.

Abstracts: Authors wishing to have abstracts of their papers appear in ABSTRACTS OF TUBERCULOSIS will facilitate their publication by sending concise abstracts or reprints to the Editor.

Reprints: Fifty reprints without covers of articles will be furnished to authors free of charge when requested in advance. A table showing cost of additional reprints, with an order blank is submitted with proofs to the authors.

Advertising: Rates will be furnished by THE AMERICAN REVIEW OF TUBERCULOSIS, 1790 Broadway, New York 19, N. Y., on request.

The publishers reserve the right to decline any advertising submitted and to censor all copy.

Single Copies: The price of single copies of this number of THE AMERICAN REVIEW OF TUBERCULOSIS is one dollar postpaid.

Vogt, J. H. Erythema nodosum, 128
Vole tubercle bacilli, 161
Volume, Pulmonary, receptors, 175

Waksman, S. A. See Geiger, W. B., *et al.*, 168
Walker, W. H. See Thomson, K. J., *et al.*, 73
Wallace, H. J. Tuberculous lymph nodes, 50
Wallach, K., and Zeman, F. D. Pneumonia in aged, 72
Wallgren, A. Treatment of primary tuberculosis, 15
Walter, A. M., Schenkein, E. L., and Sutliff, W. D. Antibodies against pneumococci, 71
War, prisoners of, Tuberculosis among, 10, 11
Warren, J. A. See Merrill, A. J., *et al.*, 126
Weber, F. J. Global distribution of tuberculosis, 2
Weil, J., and Bernard, E. Duration of pneumothorax, 142
Welch, H. J. See Brantigan, O. C., *et al.*, 37
Welding hazards, 104
Wet lung, Traumatic, 98
White, M. L., Jr., and Minor, G. R. Typhoid and salmonella infections, 87
White, M. L., and Valle, A. B. Penicillin in pulmonary resections, 39
Whorton, C. M., and Chapman, C. B. Miliary tuberculosis, 44
Wiles, F. J. See Dormer, B. A., *et al.*, 80
Wiley, M. M. See Zintel, H. A., *et al.*, 40
Williston, Elizabeth H., and Youmans, G. P. Streptomycin resistant strains of tubercle bacilli, 168
Wilson, D. A., and Baker, H. Experimental pulmonary collapse, 92
Wine, M. B. See Hampton, S. F., *et al.*, 107
Winkenwerder, W. L. Asthma in Southwest Pacific, 105
Wood, F. C., and Hodes, P. J. Tropical eosinophilia, 79
Woolley, J. S. See Bobrowitz, I. D., *et al.*, 112
Workmen's compensation for silicosis, 103
Wounds, Chest, 126
——, ——, penetrating, Circulation in, 126
——, Thoracic, 126
Wright, D. O., and Gold, E. M. Loeffler's syndrome, 78

Xalabarder, D. Neuromuscular system of lung, 87
X-ray appearance of draining bronchi, 27
—— diagnosis in bronchiectasis, 113
—— findings, Abnormal, in surveys, 133
—— generator, 6
—— peculiarities in mental deficiency, 21
—— surveys, mass, Nontuberculous lesions in, 69

Yates, J. Acid-fast organisms in gastric contents, 43
Yegian, D., Budd, V., and Middlebrook, G. Biological changes in *M. ranae*, 60
Yerushalmy, J. Tuberculosis mortality in nonwhites, 3
——, ——., and Moriyama, I. M. Tuberculosis mortality, U. S. A. 1944, 3
Youmans, G. P. Growth rate of tubercle bacilli, 56
——, ——. ——., and Williston, Elizabeth H. Streptomycin resistant strains of tubercle bacilli, 168
Young, D. Upper respiratory infections, 180

Zeman, F. D., and Wallach, K. Pneumonia in aged, 72
Zintel, H. A., Flippin, H. E., Nichols, A. C., Wiley, M. M., and Rhoads, J. E. Pharmacology of streptomycin, 40
Zwerling, H. B., and High, R. H. Pulmonary calcifications and age, 178
——, ——. ——., —— Palmer, C. E. Pulmonary calcifications, 177
——, ——. ——. See High, R. H., *et al.*, 178

ALUM ROCK SANATORIUM

SAN JOSE, CALIFORNIA

Telephone: MAYFAIR 321

A non-profit Sanatorium for the treatment of Tuberculosis and other diseases of the chest. Located in the eastern foothills overlooking Santa Clara Valley.

Medical Director
Buford H. Wardrip, M.D.
Associate Medical Director
C. G. Scarborough, M.D.

Consultants:

Harold Guyon Trimble, M.D., Oakland
Cabot Brown, M.D., San Francisco
J. Lloyd Eaton, M.D., Oakland
Glenroy N. Pierce, M.D., San Francisco
Gerald L. Crenshaw, M.D., Oakland
Ina Gourley, M.D., Oakland

The Original is Worth $35.00

Doctors Are Christmas Seal Collectors

A hobby in which many members of the medical profession find relaxation. You may indulge at home or office — any place you have the small kit of necessary material. Inexpensive too — a collection of Christmas Seals finds a ready market.

Start now by writing the Collectors Service,
National Tuberculosis Association, 1790 Broadway, New York 19, N. Y.

NOTICE TO SUBSCRIBERS AND CONTRIBUTORS

THE AMERICAN REVIEW OF TUBERCULOSIS is published by the National Tuberculosis Association and issued monthly about the 1st of the month. THE AMERICAN REVIEW OF TUBERCULOSIS is the Official Journal of the American Trudeau Society. A volume includes six numbers and begins with the January and July numbers.

Subscriptions: The subscription price of the REVIEW is $8.00 for the calendar year. Checks should be made payable to Collier Platt, Treasurer.

Character of the Review: The REVIEW consists of two main parts, namely, (1) original articles and (2) abstracts. The original part is published monthly, its size depending upon the amount of manuscript in hand. Abstracts will appear as the amount of material warrants. Each part will be paged separately, to permit permanent separate binding upon the completion of a volume.

Manuscripts. The REVIEW invites the submission of manuscripts on any phase of tuberculosis and related subjects of interest to medical practitioners and students and workers in tuberculosis and public health. Articles are accepted for publication with the understanding that they are contributed solely to THE AMERICAN REVIEW OF TUBERCULOSIS.

Manuscripts should be sent to the office of the Editor, Dr. Max Pinner, 364 Fourteenth St., Oakland 12, California. They should be in English, typewritten on one side of the page only and with wide spacing and margins. They should be mailed flat and transmitted by first-class mail with postage for return if not available. Authors should exercise particular care in the preparation, notation and description of figures, charts and tables.

The publishers will not be responsible for manuscripts, illustrations, etc., lost in transit. In order to save expense for authors' corrections in proof, manuscript should be carefully revised by the author before submission.

Abstracts: Authors wishing to have abstracts of their papers appear in ABSTRACTS OF TUBERCULOSIS will facilitate their publication by sending concise abstracts or reprints to the Editor.

Reprints: Fifty reprints without covers of articles will be furnished to authors free of charge when requested in advance. A table showing cost of additional reprints, with an order blank is submitted with proofs to the authors.

Advertising: Rates will be furnished by THE AMERICAN REVIEW OF TUBERCULOSIS, 1790 Broadway, New York 19, N. Y., on request.

The publishers reserve the right to decline any advertising submitted and to censor all copy.

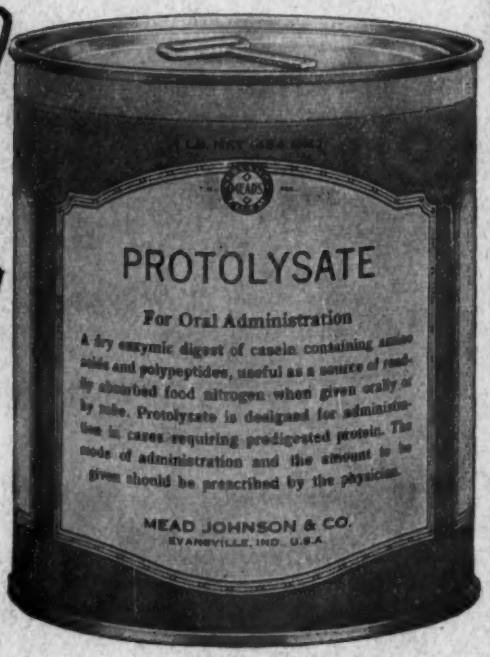
Single Copies: The price of single copies of this number of THE AMERICAN REVIEW OF TUBERCULOSIS is one dollar postpaid.

1000 cc. flasks
500 cc. flasks
125 cc. flasks
for hospitals

1 LITER (1000 cc.)
AMIGEN 5%
IN 5% DEXTROSE SOLUTION
WARNING. Do not use if the solution is cloudy or if sediment is present. The contents of this bottle must not be used for more than one infusion. For general, keep the unopened bottle in a cool place.
Test No.
MEAD JOHNSON & CO.
EVANSVILLE, IND., U.S.A.

COUNCIL-ACCEPTED
AMIGEN
for parenteral administration of amino acids

The function of Amigen and Protolysate is to supply the amino acids essential for nutrition. Both can be given in place of protein when protein cannot be eaten or digested, or in addition to protein when the protein intake is insufficient. Administered in adequate amounts, they prevent wastage of protein, restore previous losses, or build up new body protein.


PROTOLYSATE
For Oral Administration
A dry enzymic digest of casein containing amino acids and polypeptides, useful as a source of readily absorbed food nitrogen when given orally or by tube. Protolysate is designed for administration in cases requiring predigested protein. The mode of administration and the amount to be given should be prescribed by the physician.
MEAD JOHNSON & CO.
EVANSVILLE, IND. U.S.A.

1 lb. cans at drug stores

COUNCIL-ACCEPTED
PROTOLYSATE
for oral and tube feeding of amino acids

Like Amigen, Protolysate is an enzymic digest of casein and consists of amino acids and polypeptides. Like Amigen, Protolysate supplies the nitrogen essential for maintenance, repair and growth.

Unlike Amigen, which may be employed both orally and parenterally, Protolysate is designed only for oral use.

MEAD JOHNSON & CO., EVANSVILLE 21, INDIANA

There is no shortage now of AMIGEN for parenteral use. There is no shortage now of PROTOLYSATE for oral use.

ate
ial
ce
en
in
nt.
ts,
re
ly